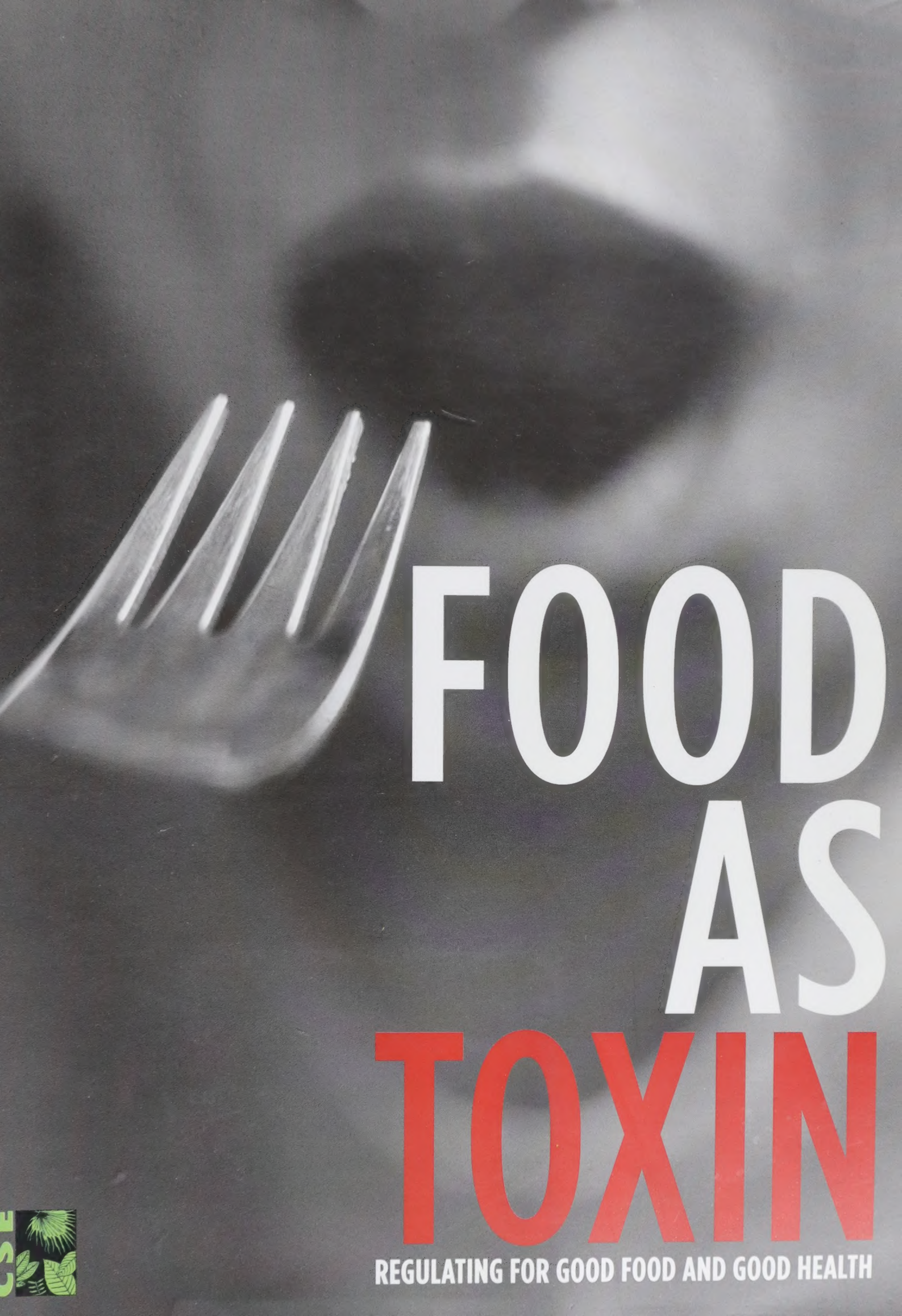

FOOD AS TOXIN

REGULATING FOR GOOD FOOD AND GOOD HEALTH

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FOOD AS TOXIN

REGULATING FOR GOOD FOOD AND GOOD HEALTH

CENTRE FOR SCIENCE AND ENVIRONMENT

NEW DELHI

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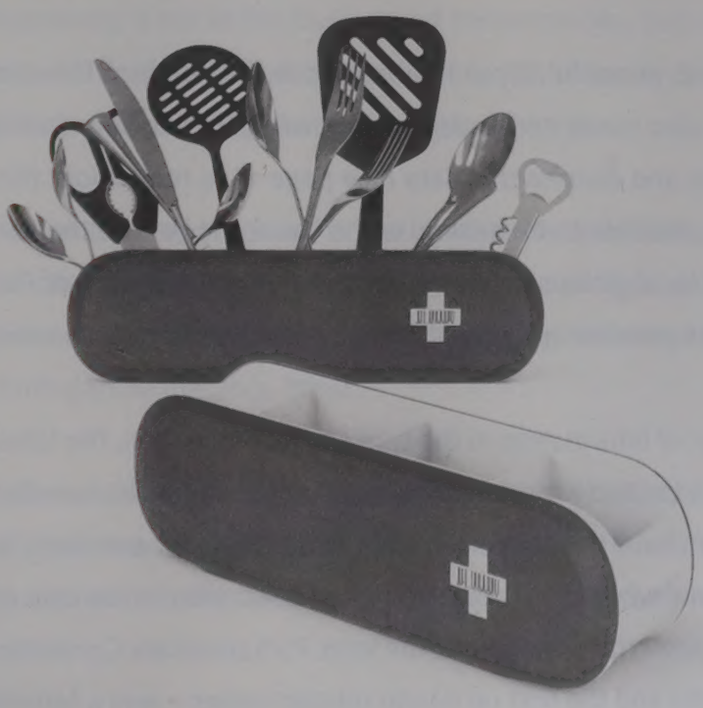


FOREWORD

When Anil Agarwal, CSE's founding director, decided to set up a testing laboratory for toxins and environmental contaminants we could not understand his reasoning. CSE had no track record in doing analytical testing, let alone running a state-of-the-art environmental monitoring facility. We were a policy research institution, comfortable with using our information for policy change.

But Anil was convinced that this laboratory was essential. He said that we desperately needed science in the public domain that would challenge, indeed raise, critical issues of ecological security. His reasoning also stemmed from the fact that every time CSE had approached laboratories for testing contamination we had either been turned down – government and university laboratories said "this is too controversial" and private laboratories were just too expensive. As a result, even where we knew there was a problem, we could not test and therefore, could not prove that there was evidence of contamination. And so we could not successfully push for change. In 2000, the CSE Pollution Monitoring Laboratory (PML) was set up.

The first test of the laboratory came by chance. We had a letter in the post from a doctor who wrote to say that he was seeing terrible human affliction in his small village of Padre, located in the hills in Kerala. A journalist colleague was sent to investigate the issue. But this time, because we also had the facility to test the problem, we decided to get samples of blood, soil and food collected. The tests were done. The analysis showed very high levels of a pesticide called endosulfan in all samples. Why, we asked. We learnt that the cashew plantation around the village was aerially sprayed with this pesticide each year, year after year. We also learnt that the last spray was done just a few days before we collected samples.

The test result was powerful. It put information in the hands of the community. But because it was powerful, it was also contested. Indeed the powerful pesticide industry has worked overtime to dispute, discredit and dismiss this data (see page 18 to read about this vicious attack). It has taken 10 years for the pesticide to be banned in the country. I believe the ban was the result of perseverance of local groups, who have ensured that the people of Padre village get justice. But also, the ban was possible because this time there was hard data which led to change.

This is the power of information in the true sense. Since then, the laboratory has gone ahead to test pesticides in bottled water, antibiotics in honey and most recently, junk in junk food. In all the cases, the results have been received with, first, contempt and then, dismissal and nasty rebuttals. The acid test came when the laboratory tested pesticides in the cola majors – Coke and Pepsi – and was faced with powerful opponents. The Joint Parliamentary Committee – the fourth JPC of independent India and the first on health related issues – was a formidable challenge for us. The

attack was on our ability to test 'complex' cola problems. The slur was that we were not competent enough – the CSE laboratory had simply got it wrong. But at the end, after substantial discussion and retesting of samples by government laboratories, the JPC concluded, "the Committee finds that the CSE findings are correct on the presence of pesticide residues in carbonated water. The Committee appreciates the whistle blowing act of CSE in alerting the nation to an issue with major implications to food safety, policy formulation, regulatory framework and human and environmental health."

This was an endorsement of our work. More importantly, it pointed to the importance of research to drive policy and practice change. There is no doubt that the CSE Pollution Monitoring Laboratory has made a huge contribution to bring issues of food safety and toxins to public knowledge in India. The challenge is massive. We need to develop, grow in productivity and economic prosperity. But how do we do this without compromising on health and nutrition security? The CSE laboratory, through each study, has shown convincingly that the options are to find leapfrog solutions – to cut the toxin treadmill and not to first contaminate our food, our environment and poison our bodies.

The laboratory is always asked two things. First, if it is certified for testing. The answer is yes – ISO 9001/2008 – for quality management systems. But the more important accreditation it has is from the public scrutiny of its research. It is clear to us that government accreditation systems are both cumbersome and open to abuse. We also know that many laboratories that have this accreditation do shoddy and compromised research. We have therefore decided that the best certification comes from putting the data in the public domain, so that it can be critiqued and checked by all. This public disclosure is our test.

Secondly, we are asked why we do not share the results of our tests with companies before release. The fact is that CSE laboratory is not in the business of consultancies, but of policy change. It is therefore, logical for us to make the research public and to use this research to demand changes in policies and practices, particularly regulatory changes.

Our work will continue. What needs to be appreciated is that a relatively small laboratory has made such an enormous difference in changing regulatory systems and food and toxin policies in the country. But equally, we need to ask why the existing paraphernalia of government laboratories and research institutions are failing to do just this. Why?

This needs to be debated and discussed. The country needs science for ecological security. It needs science for public policy. The CSE laboratory and the research it has conducted has played a seminal role is doing just this. Now we need more institutions to join: to make the future change possible.

Sunita Narain

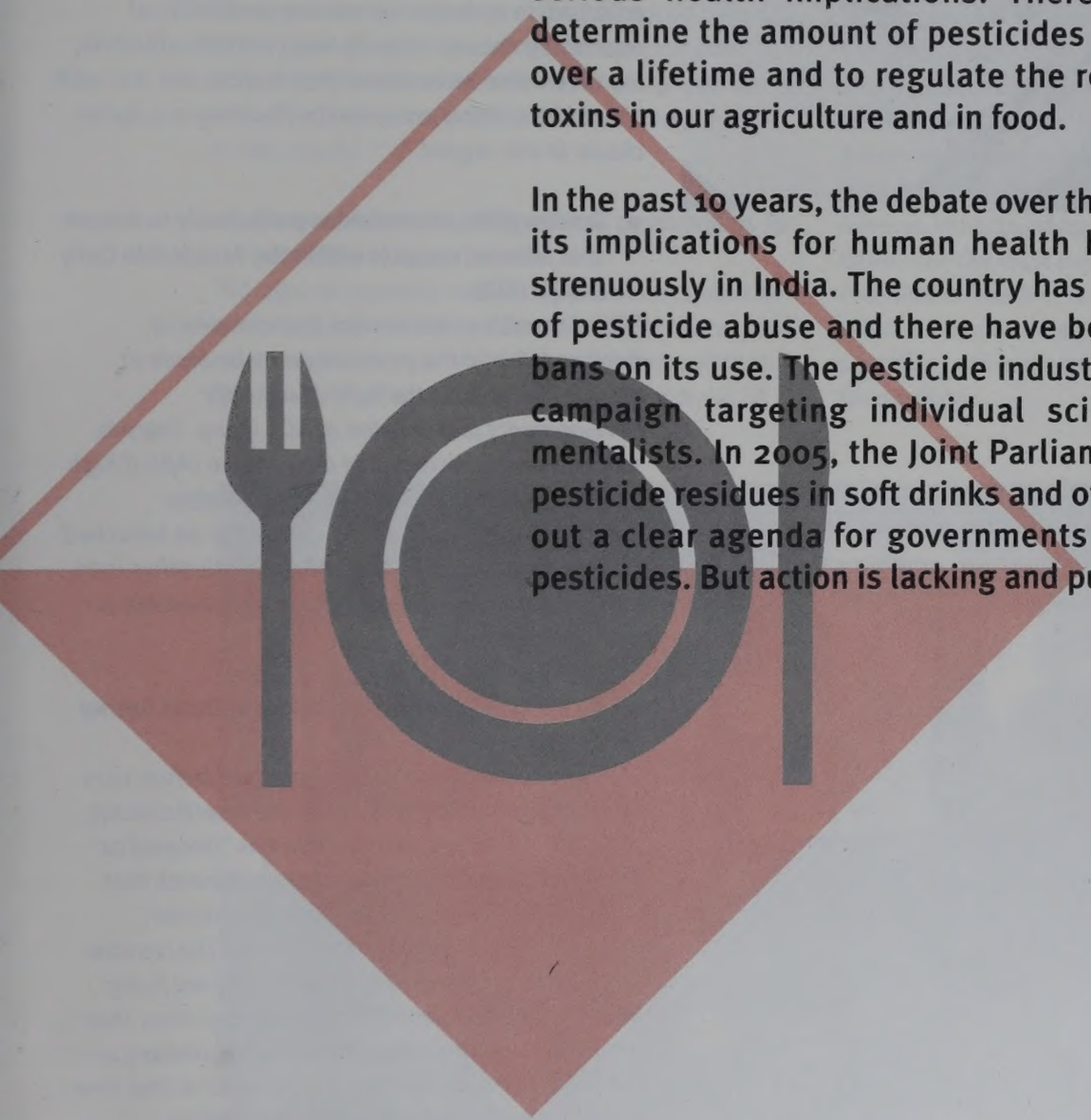
TOXINS IN AND AROUND US

1

"All substances are poisons; the right dose differentiates a poison and a remedy." Modern food regulation is about determining what is that right dose in our daily diet.

We know pesticides are toxins, chemicals designed to kill insects and other creatures at low doses. Unless human exposure to toxins is regulated and minimised, there are serious health implications. Therefore, it is critical to determine the amount of pesticides we can be exposed to over a lifetime and to regulate the registration and use of toxins in our agriculture and in food.

In the past 10 years, the debate over the use of pesticides and its implications for human health has been fought over strenuously in India. The country has learnt about the cases of pesticide abuse and there have been calls for stringent bans on its use. The pesticide industry launched a virulent campaign targeting individual scientists and environmentalists. In 2005, the Joint Parliamentary Committee on pesticide residues in soft drinks and other beverages had set out a clear agenda for governments to ensure safe use of pesticides. But action is lacking and public health is at risk.



1.1 CONTAMINATION IN FOOD

1. What did the Joint Parliamentary Committee (JPC) direct the government to do to regulate safe use of pesticides?[1]

- **Register a pesticide only after Maximum Residue Limit (MRL) has been fixed. Amend the insecticides Act, 1968 to include a provision for making this mandatory.**

"The Committee were anguished to note that pesticides were being registered by the Registration Committee even when no MRLs had been fixed. It is only after the CSE came out with their report on presence of certain pesticides in the bottled water... that a decision was taken by the Ministry of Agriculture... to discontinue this practice. The Committee desires that this should now be strictly enforced. In order to rule out any possibility of registering the pesticide by way of notification/rule, the Committee recommend that Insecticides Act 1968 should be suitably amended by inserting a suitable clause in this regard."

- **Review MRLs of pesticides periodically to ensure that allowed usage is within the Acceptable Daily Intake (ADI).**

"The Committee also desires that a review of existing MRLs of the pesticides may be made at regular intervals, in the light of scientific developments and revision of ADI, if any. There is scope to exceed acceptable daily intake (ADI) if high MRLs have been set because ADI is a safety milestone and should not be allowed to be breached and the basic purpose of setting realistic MRLs is to ensure that we remain well within allocated ADI for that pesticide."

- **Fix MRLs of 'deemed' pesticides without further delay.**

"The pesticides which were being used before 1971 i.e. prior to coming into force of the Insecticide Act, 1968 and rules 1971 were included as "deemed as registered pesticides". The Committee notes that many of the MRLs of the "deemed registered pesticides" have not been fixed so far. The reasons given by the Ministry of Agriculture, for not fixing MRLs for deemed pesticides, that at that time, their usage data was not complete, is not convincing as the Committee feel that even if this data at that time was not complete or available, Registration Committee should have asked the manufacturers of

these pesticides to supply the data and fix their MRLs. Though many of the deemed pesticides are already phased out, the Committee desires that MRLs of deemed pesticides which are still in use may be fixed without any further delay."

- **Collect information about the use of banned pesticides and check this practice.**

"The Committee also find that neither the Ministry of Agriculture nor Ministry of Health & Family Welfare have any data about the usage of banned pesticides in the States since inception. The Committee wonder as to how the Ministry of Agriculture which have made claims before the Committee towards Integrated Pest Control Programme are monitoring the very use of pesticides in the absence of such vital data. It does speak volumes about the apathetic attitude of the various functionaries. The Committee however desire that Ministry of Health and Family Welfare in coordination with the Ministry of Agriculture should impress upon the State Governments the imperative need of strictly adhering to the guidelines for usage of DDT, Lindane and other restricted pesticides for health programmes only. The farmers too need to be educated properly in this regard."

- **Do an annual monitoring of food commodities for pesticide residues.**

"No agency regularly monitors pesticide residues in market samples or undertakes diet basket surveys to assess actual exposure of consumers from pesticide residues in food or water and project health risk, if any. Such activity comes under the purview of Ministry of Health but no comprehensive regular monitoring programme is being conducted in the country. The Committee feel that such monitoring of food commodities requires to be done extensively and on yearly basis."

2. *Why do we have to accept pesticides in our food? Why should we have to spend money to regulate pesticide residues in our food?*

What is 'safe' means calculating what we eat, how much we eat and how much pesticide is allowed in the food we eat. Therefore, the food basket is also the pesticide basket. Put simply, we have to eat pesticides because we have to eat food for nutrition, but we must not exceed our quota because that would be deadly. Call this the nutrition and poison trade-off. As long as we cannot wish away pesticide use, it is imperative that this evil is prudently handled.

3. *How is food safety defined?*

Calculating our exposure – what we can eat safely – requires regulators to understand what we eat and how much. It is a combination of health and toxicological science – to determine the level at which the chemical does not have any adverse impacts; of agricultural science – to determine the least possible pesticide residue on the crop that is feasible; and of nutrition and dietary science – to calculate what we eat and how much pesticides will we ingest (see Box: *A guidebook to food safety*).

What does 'Acceptable Daily Intake' (ADI) mean?

ADI is the safety touchstone for regulating pesticides in food commodities. Problems posed by pesticides led to the global scientific community developing the concept of ADI. The ADI principle refers to the amount of a pesticide we can ingest, daily, over a lifetime, without damaging our health. It is expressed in relation to bodyweight, so that safety levels of pesticides for adults and children can be calculated separately. Regulators across the world make sure that pesticide exposure of the population never exceeds the ADI. The following process is used to develop ADI: toxicity tests are done on animals, which are given different amounts of a pesticide and checked for a range of toxic effects, including birth defects, cancer, reproductive changes, neurotoxicity and harm to organs such as the kidney or liver. The idea is to determine that limit till where a pesticide cannot cause harm: this is called NOAEL, or 'No Observable Adverse Effect Level'. Sometimes, it is not possible to deduce this number. In such cases the safety mark is established at that point where the first sign of adverse effect appears. This is called LOAEL, or 'Lowest Observable Adverse Effect Level'.

Both these measures indicate the long-term effect on health, or chronic toxicity. But pesticides are highly poisonous; a single dose can be lethal. To tackle such circumstances, global agencies also establish safety limits for acute toxicity or exposure in the short term. Thus, the JMPR and the USEPA set what is called the Acute Reference Dose (ARfD), the maximum pesticide residue that can be allowed for one-time exposure. Acute toxicity is typically calculated from Lethal Dose 50 (LD-50) – literally a potent quantity of pesticide that can kill 50 per c of test animals either through ingestion or through cont with skin.

Scientists then extrapolate animal toxicity data on huma They adjust it downward usually by a factor of 100: a divis factor of 10 is used to allow for the possibility that humans more sensitive; a further division factor of 10 is used to all for differences between individual humans. Nowadays, ther increasing concern that this safety factor leaves infants a children vulnerable to pesticide toxicity attack. In the US, instance, health activists want a further safety factor of 10 children – so that the toxicity data is adjusted downward b factor of 1,000, especially for organophosphate class pesticides.

Today, the use of ADI is a crucial tool to mana pesticide risk.

What does Maximum Residue Limit (MRL) mean?

Regulating pesticides on the ground revolves around regulatory yardstick called the MRL – or Maximum Residue Li – of a pesticide in a food commodity. It is a legally enforceal standard. It is set for a single pesticide, on all the different fo items in which that pesticide's residues are likely to be fou MRL is determined via supervised field trials of pesticides crops. The intention is to arrive at practicable ways to minimi the pesticide residue in crops. Agricultural scientists work determine the best agricultural practice and recommend t crops for which a pesticide should be used, and how it should used. Globally, if pesticide residue in a food commodity excee its MRL, then that food is legally considered adulterated, a penalties can be imposed.

What is the relationship between MRL and ADI?

The relationship between the MRL and ADI is the one that rea determines safety.

Exposure to a pesticide occurs primarily via what we ea Regulators know the residue level legally allowed for pesticide on a food commodity, its MRL. Having figured out ho much of that food we eat daily, preferably at the national lev they multiply the two quantities to arrive at the pesticide inta we can be legally exposed to. For instance, Indian MRL for t pesticide monocrotophos in rice is 0.025 mg/kg. Indian diet f rice is 209 gms/day. Thus, exposure to this pesticide through ri is 0.005 mg/day.

In this way, after multiplying the MRL of a pesticide various food commodities with the respective amount commodities we eat and summing them up, we arrive at th Theoretical Maximum Daily Intake (TMDI). In no case should th TMDI of a pesticide calculated using its MRL exceed the ADI that pesticide. This is the bottom line of the safety mechanis set across the world for regulating pesticides.

If TMDI is above ADI, then all the factors involved in settin

Ls is reviewed and reworked. To bring the TMDI below ADI, trictions can be placed on the use of pesticides in food mmodities so that the number of commodities through which posure happens is reduced. Conversely, if permission is given the pesticide to be used on a new crop, then the entire TMDI ocess is reworked and the value of MRL of the pesticide on e existing crops is reduced.

In cases where an ADI cannot be established, for example in se of a very toxic pesticide, MRLs are set at 'no detection' levels here, no residue is allowed on any food commodity. Finally, the RLs are also adjusted to build in exposures from other pathways ch as water and air, always keeping ADI in mind.

ow do regulators know how much we eat? Don't diets ry across the world?

e first guidelines for predicting dietary intake were prepared the Global Environmental Monitoring System (GEMS/Food) in 89, which were later revised in 1997. Global regulators divide e world into five regional diets (Middle Eastern, Far Eastern, rican, Latin American, and European) to estimate what people t. India falls broadly in the Far Eastern regional diet, which is e and cereal-based.

The data used to compile these global diet charts is put gether using the average food consumption data given in O's food balance sheets. Although the food consumption data rived from such food balance sheets could be uncertain, they present the best available source of data for international mparison and are adequate for predicting pesticide residue take. But because such estimates are inadequate for national olicy making, it is important for a nation to develop its own tional dietary model to estimate exposure.

ow do governments improve the estimations of how uch we eat?

s food consumption patterns vary considerably from country country and even within a country, individual nations have to so estimate their own consumption patterns. They do this rough improvements in their diet studies.

The first level of estimation is based on an average per pita diet: the TMDI. The second and more accurate level is by calculating the exposure based not on the set MRL, but on the actual residues found in the food – the estimated daily intake (EDI). The third and most accurate level is by measuring the intake on cooked food – the Total Diet Study.

Are countries able to effectively control pesticide contamination in food by using ADI and MRL?

Countries following this process have been able to combat health risks from pesticides. Here are the examples from some countries:

The US has assigned clear responsibilities to two nodal agencies – the USEPA and the US Food and Drug Administration (USFDA). The former, a standard-setting body, is entrusted with registering a pesticide for use. Before registration, it establishes ADI – what it calls as Chronic Reference Dose (CRfD) – for that pesticide and sets MRLs for residues on food commodities. It makes sure exposure is well below ADI. The USFDA is the enforcing agency. It collects samples of food – raw and processed – and analyses them for pesticide residues, checking them against the USEPA-set MRLs. If 'illegal' residues are found in either kind of samples, the USFDA invokes various sanctions, such as a seizure or injunction. As a huge number of pesticides are used in the US, the USFDA uses a multi-residue test method that can detect up to 400 different pesticides in a food commodity. The process the US follows has been able to significantly reduce pesticide exposure.

The EU's process is quite similar to what the US does. Here, the MRL is legally enforceable; non-compliance leads normally to legal proceedings against the supplier. Of late, the EU's Working Group on Pesticide Residues has adopted a policy of "naming and shaming" suppliers and wholesale agencies whose food samples were found excessively contaminated. The most recent annual report of the pesticide residue monitoring programme, published by the Food and Veterinary Office of the European Commission states that out of the 46,000 samples of fruits, vegetables and cereals tested by the member countries, only 4 per cent of the samples exceeded MRL, that too by a mere 1.3-9.1 per cent. In the EU, too, the exposure is well within ADI.

In Australia, the ANZFA monitors pesticide residues in foods. According to its dietary exposure report for 2002, the pesticide exposure for different ages and genders were below 16 per cent of the ADIs of all pesticides. All the exposures were found to be well within applicable health standards.

In Canada, too, enforcement works. The Canadian Food Inspection Agency (CFIA) tests thousands of samples annually to ensure MRLs are not exceeded. A four-year study from 1994-1998 tested 44,378 products – domestic and imported – and found that 98 per cent of the samples were in compliance. About 93 per cent of processed foods and 75 per cent of fresh samples contained absolutely no residues at all. A five-year study of water wells found that 99.9 per cent of the tested wells met government standards for pesticide residues in water.

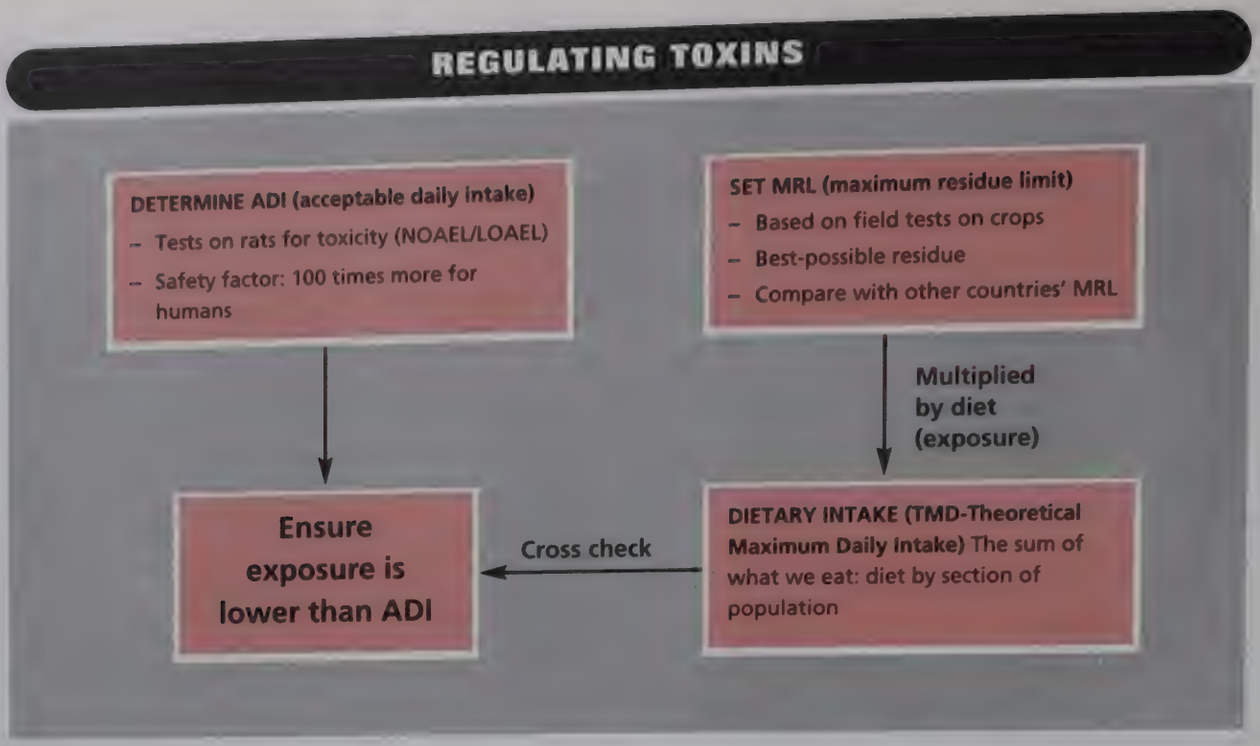
Calculate your daily dose of malathion

Calculate it as per JMPR (WHO/FAO) ADI:

- 0.3 mg/day x 60 kg of bodyweight = 18 mg/ day for you
- 0.3 mg/day x 10 kg of bodyweight = 3 mg/day for a child

Calculate it as per USEPA ADI:

- 0.024 mg/day x 60 kg of bodyweight = 1.44 mg/day for you
- 0.024 mg/day x 10 kg of bodyweight = 0.24 mg/day for your child


REGULATING TOXINS
DETERMINE ADI (acceptable daily intake)
– Tests on rats for toxicity (NOAEL/LOAEL)
– Safety factor: 100 times more for humans
SET MRL (maximum residue limit)
– Based on field tests on crops
– Best-possible residue
– Compare with other countries' MRL
Multiplied by diet (exposure)
Ensure exposure is lower than ADI
Cross check
DIETARY INTAKE (TMD-Theoretical Maximum Daily Intake) The sum of what we eat: diet by section of population

4. *What are the global systems that regulate food safety?*

The world awoke to pesticide contamination of food years ago. In 1953, a resolution at the World Health Assembly expressed concern about "the increasing use of various chemical substances in food". This inaugurated a process that led, two years later, to the formation of 'Joint Food and Agricultural Organization (FAO) and World Health Organization (WHO) Expert Committee on Food Additives' (JECFA). The JECFA's initial mandate was to review and set safety standards only for all food additives — it defined additives as "non-nutritive substances added intentionally to food, generally in small quantities to improve its appearance, flavour, texture or storage properties". Later, its mandate was broadened to include substances "unintentionally" introduced into food, such as pesticides.

Then in 1961, this latter function was turned over to another body. The JMPR, or the 'Joint FAO/WHO Meeting on Pesticide Residues', which provides scientific advice to the Codex Alimentarius Commission (a wing of the United Nations to oversee fair international trade practices in agricultural commodities) on regulating pesticide residues in global food trade, was established to direct global food standards for pesticide contamination. Today, while these agencies set the global framework for safety, nations individually develop their own standards.

5. *How are pesticides regulated in India?*

Till recently, two legislations regulated pesticides in India – the Insecticides Act, 1968, which is implemented by the Union ministry of agriculture; and the Prevention of Food Adulteration (PFA) Act, 1954, which is implemented by the Union ministry of health and family welfare. The Insecticides Act, 1968 is enforced in part by the Central Insecticides Board (CIB) and the Registration Committee (RC) formed under it. The over 25-member strong CIB is headed by the director general of health services and advises on matters related to administering the Insecticides Act. The RC registers pesticides for use in India and for export. It is supposed to do this after satisfying itself about a pesticide's efficacy and safety to human beings, animals and the environment; relevant data to this end are collected from companies.

In the past few years, some changes have come into force. In 2006, the Food Safety and Standards Act was enacted and the Food Safety and Standards Authority was given the mandate to regulate pesticides in food. The government has also introduced the Pesticide Management Bill, 2008 in the Parliament, which is awaiting clearance.

6. *Who sets the MRLs for pesticides before registration in India?*

Till 2008, MRLs were set by the Directorate General of Health Services on the basis of recommendations made by the Pesticide Residues Sub-Committee of the Central Committee for Food Standards (CCFS), under the Union ministry of health. The CCFS would meet once or twice a year, and standards would be set on the basis of information supplied by government research institutions and companies. The limit set by this sub-committee would be

provided to the CIB or Central Insecticides Board, which would then register the pesticide and mandate the crops for which the pesticide would be allowed. This system has undergone changes, which are adding to policy confusion and compromising health.

In a reply (dated April 25, 2011) to an RTI application, the ministry of agriculture has said clearly that the tolerance limit (MRL) of pesticides is being set under the PFA Act, 1954 and being implemented through the ministry of health and family welfare.[2]

But as of 2006, the mandate for setting pesticide limits has moved to the new food safety authority. The FSSAI's Scientific Panel on Pesticides and Antibiotic Residues had its first meeting on December 17, 2009, chaired by Debabrata Kanungo, additional director general, Directorate General of Health Services. The meeting agreed that the Panel would provide MRLs within a period of three months after it received the complete set of information from the pesticide manufacturing company.

No meeting of the Panel took place in 2010. The minutes of the second meeting of the Panel have not been provided (possibly did not happen) in spite of RTI queries. The third meeting was held in August 2011, when the matter of fixing MRLs was postponed to the next meeting.[3]

At the fourth meeting, a proforma for "submitting data on residues and toxicity by the applicant seeking registration of new pesticide" was submitted. In addition, the submissions received from the ministry of agriculture for fixing MRLs of six pesticides on different food products were examined. These were:

- Orthosulfamuron on paddy
- Pyradalyl on chilli
- Deltamethrin on chilli
- Propragite on brinjal
- Fenazaquin on brinjal and tomato
- Lambdachhalothrin on grapes
- Ethephone on pomegranate

The Panel listed all the documents, which were incomplete or unavailable to set the MRLs, and then went ahead and set limits for five of the six pesticides.[4]

At the fifth meeting on October 2011, the limit for deltamethrin on chilli was revised. The MRL for orthosulfamuron on paddy was also discussed. The panel recommended the MRL as 0.1 mg/kg body weight. No information is provided on how this MRL was fixed.[5]

At the sixth meeting on October 28, 2011, the MRL for imidaclorprid in chilli was fixed as 0.3 mg/kg. No further information is again provided to suggest the basis of this number and how this was established.[6]

At the eighth meeting, held on December 17, 2011, MRL was fixed for the following:

- Flubendamide for brinjal and Bengal gram
- Thaimethoxam for tomato and cumin
- Fenoxaprop-p-ethyl for cotton seed
- Betacyfluthrin for okra and brinjal
- Imidcloprid for brinjal

The MRL was not fixed for quizalofop ethyl for onion and black gram.[7] But interestingly, this pesticide is listed as a registered pesticide on the website of the Central Insecticides Board.[8] Therefore, clearly, there is no compliance with the directive that pesticides can only be set after MRLs have been decided.

7. Is the government in compliance with JPC directions in setting of MRLs for every pesticide before registration?

No. Under the Food Safety and Standards Act, 2006, no article of food shall contain pesticide residues in excess of the MRL specified under the law.

As per the amended Food Safety and Standards (Contaminants, Toxins and Residues) Regulation 2011, there are a total of 149 registered pesticides for which MRL has been specified in different food items.

However, as per the CIB and the Registration Committee (RC) under the ministry of agriculture, there are a total of 234 pesticides which have been registered for use in the country. Therefore, there is a clear difference between the two lists.

The ministry, in its RTI reply, admits that there are 10 pesticides for which no MRLs have been fixed, but these have been registered.[9]

Furthermore, the draft notifications on pesticide residues in food articles (GSR 261 (E) dated 20.4.2009, GSR 328 (E) dated 15.5.2009 and GSR 264 (E) dated 30.3.2011) remain draft notifications and have not been issued. It is not clear if these pesticides have been registered.

The government is clearly in breach of the directions of the Joint Parliamentary Committee.

8. Is the government in compliance with JPC directions on fixing MRLs for 'deemed' pesticides?

No. At the third meeting of the FSSAI's Scientific Panel on Pesticides and Antibiotic Residues held in August 2011, the JPC direction to fix MRLs for 'deemed' pesticides was discussed for the first time. It was agreed that there are a total of 71 pesticides in this category. But these also include pesticides which have been banned. The decision was to discuss this matter at a later date. Since then, the issue has been listed in the agenda, but no decision has been taken.

9. *What is the legislative procedure to ensure that no pesticide can be registered without the setting of MRL?*

The JPC had noted that there is also no legislative provision to link pesticide registration to setting MRLs. The Insecticides Act mandates registration, but the PFA mandates MRLs. Lack of coordination has led to huge irregularities in overall pesticide regulations.

In mid-2003, for the first time, an effort was made to ensure that registration of a pesticide by the CIB would simultaneously require that the ministry of health fixed the MRL for that pesticide on different foods. At an inter-ministerial meeting held on June 2, 2003 under the chairpersonship of the secretary (agriculture and cooperation), it was decided that pesticides would not be registered without fixing the MRL.

But the pesticide registration committee refused to accept this directive. In its 238th meeting held on September 12, 2003 the Registration Committee (RC) discussed the decisions reached by the inter-ministerial meeting. The committee decided that it would forward the data required for setting MRLs to the ministry of health and family welfare and wait for a period of four weeks. The RC also decided that if in any event, no response on the MRLs was received from the health ministry within the stipulated period, the committee will go ahead and consider the pesticide for registration despite MRLs not being set. It reiterated its decision that the "fixation of MRL subsequent to supply of requisite data is a separate issue and linking this with registration will unnecessary delay registration".

In a meeting held on March 31, 2004 under the chairpersonship of the additional secretary, ministry of agriculture, to discuss procedure for fixing MRLs for pesticides, it was again reiterated that registration would only be done after the ministry of health fixes MRLs. But the RC has continued to ignore these directions. An analysis showed that the RC went on to register pesticides without fixing MRLs in its subsequent meetings on April 2, 2004; May 27, 2004; and July 2, 2004 (243rd, 244th and 245th meetings of the committee, respectively).

The RC has consistently rejected the need for MRL standards being set before the registration of the pesticides on the ground that this will unnecessarily delay the registration process. The process followed by the committee is to send the information to the ministry of health for fixation of MRLs. But often the data is incomplete and decisions are delayed. The registration committee is able to bypass the provisions by registering the pesticide without setting an MRL.

Currently, the system is run on an ad hoc basis through correspondence between the concerned departments, with little information available in the public domain about its integrity. The ministry of agriculture sends information to the FSSAI about the pesticides that need MRLs. The FSSAI scientific panel meets and sets the MRLs; this information is recorded in its minutes (which are not in the public domain). The FSSAI scientific panel writes and informs the ministry of agriculture of its decision and the Central Insecticides Board then issues a registration letter to the company. However, the General Statutory Rule (GSR), which notifies the pesticide residue, is not issued before the registration is given. There is no data available on the website of FSSAI or CIB about the MRLs set for pesticides.

10. *Has the government amended the insecticides Act 1968 to include provisions to make the fixing of MRLs mandatory, as directed by the JPC?*

Not yet. In the Pesticide Management Bill 2008, which has been tabled in Parliament, there is an explicit provision:

'12(5): No pesticide shall be registered for import or manufacturing unless its tolerance limit are specified for its residues on crops and commodities under the Food Safety and Standards Act 2006.'

Once this Bill is passed, then regulation of pesticides will also move a small step ahead.

11. *Is an MRL set for all the crops for which the pesticide is registered to be used upon?*

No. This is a major lacuna in the system. The RC registers a specific pesticide, but the MRL is set for pesticide use in a particular crop. The use in crops is only a labeling requirement as the pesticide is recommended for use on a particular crop. This is not reflected in the registration data or anywhere on the site of the Central Insecticides Board.

If a manufacturer wants to expand the use of the pesticide in other crops, they are required to apply for a 'change in label'. It is also required that this change of label is only done after the MRL has been set. But as this is not a legal requirement – nowhere in the Insecticides Act, 1968 or the Pesticide Management Bill, 2008 does it say that the pesticide can only be registered for specific crops and that any expansion for use in additional crops, is a change in the registration.

12. *What is the problem if a farmer uses a pesticide which is recommended to him/her, but which has no MRL set for it?*

Under the law, if residues are found on the crop of a pesticide for which there is no MRL, or allowed limit,

the crop is adulterated. Therefore, farmers are caught between two laws of the country.

Today, there is huge disparity between two authorities, one which regulates pesticides in the country (CIB) and the other which regulates residues on food commodities (formerly the Central Committee for Food Standards, or CCFS and now the Food Safety and Standards Authority). While the CIB recommends farmers to use pesticides on different crops, the CCFS does not fix the MRLs for the pesticide residues in all the crops that the farmer has been recommended to use. This leads to a gross mismatch between the MRLs set for pesticides and those pesticides recommended for use by the RC.

This leads to a very unusual situation. A farmer is 'recommended' a pesticide for a crop, and uses it for that crop. In doing so, he or she follows the law as laid down under the Insecticides Act. But if there is no MRL for that pesticide under the PFA Act; the crop cannot legally contain any pesticide residue. Now, if this farmer's crop shows residues of the 'recommended' pesticide, he would be violating PFA provisions.

This needs to be fixed by mandating that pesticides are registered for use on particular crops and only after the MRL has been set. The information of the MRL must be available on the registration information provided by CIB.

13. *Does the government conduct an annual analysis of pesticide residues in food, as directed by the JPC?*

Yes, but not really.

In 2005-2006 the Monitoring of Pesticide Residues at the National Level was started as a central sector scheme under the ministry of agriculture. The project, with some 21 laboratories across the country, is expected to analyse samples of food commodities. Its first report analysed 14,225 samples collected during the period 2009-2010. Its second report analysed 15,321 samples collected during 2010-2011.[10,11]

According to this analysis, there is little to worry about – no contamination has been found and samples above the maximum residue level are miniscule. This is in sharp contrast with the analysis available, from this source, for the period 1999 and 2000. In 1999, the analysis had found that 20 per cent vegetables were above the MRL; in 2000, 12 per cent were above. In the 2000 report, over 77 per cent packaged milk samples exceeded the MRL for HCH and 43.4 per cent exceeded the MRL for DDT.[12] A

'CLEANED' OUT: PESTICIDES DISAPPEAR FROM FOOD IN INDIA

Food commodity	% above MRL 2009-2010	% non recommended 2009-2010	% above MRL 2010-2011	% non recommended 2009-2010	% above MRL 2000
Vegetables	2	3.3	2.3	NA	11.7
Fruits	0.8	2	0.9	NA	
Packaged milk	0.9	1	Nil		50% and above
Animal feed	–	–	Nil	NA	No MRL but over 50% samples contaminated.
Spice	0.9	27	5.6	NA	
Wheat and rice	3	2.7	1.6	NA	
Fish	1.7	0.9	Nil	NA	
Black tea	Nil	1.6	Nil	NA	
Pulse	--	4.8	NA		
Meat	Nil	Nil	Nil		
Eggs	Nil	Nil	Nil		
Honey	Nil	Nil	NA	NA	
Dry fruit	Nil	Nil	NA	NA	

Sources: All India Network Project on Pesticide Residues 2010, Monitoring of Pesticide Residues at National Level, *Annual Report 2009-2010,* ministry of agriculture, *mimeo;* All India Network Project on Pesticide Residues 2011, Monitoring of Pesticide Residues at National Level, *Annual Report 2010-2011,* ministry of agriculture, *mimeo*

HOW TO STRENGTHEN INDIA'S FOOD LABORATORY INFRASTRUCTURE

This is a big ticket item; essential for an effective food safety system but also very expensive in terms of capital investment and even more difficult to operate. The experience of setting up laboratories in the country has been extremely poor. Even worse is the experience of setting up laboratories in the states, through Central assistance. In most cases, the funds paid by the Central government for hardware remain unutilized. There is no money to pay for personnel, nor a management system that drives quality and accountability in the functioning of these laboratories. Currently, according to FSSAI's own assessment, the existing Central and state laboratories are weak, defunct and non-functional in most cases.

In its draft proposals for the 12th Five Year Plan, FSSAI has proposed setting up/upgrading the following:

- Central laboratories – upgrading the Ghaziabad laboratory into the National Food Science and Risk Assessment Centre
- Setting up a new central laboratory in Mumbai and upgrading the Kolkata laboratory
- Upgrading 10 laboratories from the 72 existing state laboratories into referral labs
- Upgrading remaining 62 labs in states as district laboratories
- Setting up another 150 labs at the district level
- Setting up mobile testing laboratories

All this is estimated to cost Rs 1,200 crore, without adding the cost of personnel; another Rs 2,530 crore will be contributed by the state. It is strongly argued that the state governments do not have the interest, finances or wherewithal to fund the 'hardware' investment needed for statutory functions. Therefore, the FSSAI has also proposed the creation of a Centrally-sponsored scheme which will fund the setting up of laboratories in the states.

This proposal is indeed laudable, as the country needs serious and urgent enhancement of its capacity to test food products. But the proposal does not take into account the current problems with such capital – and hardware – intensive infrastructure creation and in particular, the funding of its operations and supervision by the states.

The proposal for laboratories does not also differentiate between the different needs of testing products for different categories of problems. The proposal is extremely hardware – focused as it assumes that all equipment will be needed at all levels.

It is important to recognise that India, even as it is growing economically, will not be able to afford the scale of laboratory infrastructure that may be required for the modern food sector. It is therefore even more important to devise strategies and policies that prevent the growth of bad food (now well classified globally, as food that is poor on nutrition) and not think of regulating it once it is widespread.

Therefore there are two distinct objectives of a food surveillance system in traditional-modern country like India.

a. The need to check basic food adulteration, which is common in unbranded food. This is easier to check and does not require expensive lab instrumentation. But it does require a system in which the tests are done and results are publicly disseminated. These laboratories should be available in different parts of the country.
b. The need to check for food safety, which requires more expensive instrumentation needed for modern contaminants, like pesticides, antibiotics, hormones etc and harmful additives. These laboratories, because of the level of their requirement of instrumentation and skilled personnel, should be located strategically.

decade later nothing has been found and virtually no samples exceed the tolerance limit set.

In addition, the food laboratories under the state food regulators and the Central Food Safety and Standards Authority also test food products for pesticide residues. However, the analysis done by these laboratories has been found to be lacking and poor in quality. In March 2012, the Delhi High Court listening to a matter on high pesticide residues in vegetables in the Delhi region, lambasted the FSSAI for claiming that there is a well-established system to monitor toxins in food products. "We find these pleadings surprising because the material brought on record shows a picture which is far from the high levels of pesticides found in vegetables in the NCR region" noted the two-judge bench. The Court then asked the ministry of agriculture and the FSSAI to submit a joint report on this matter. Now the FSSAI has asked the Central government for a massive budgetary allocation under the 12th Plan to upgrade its food laboratories. But the question is whether money alone will be enough (see Box: *How to strengthen India's food laboratory infrastructure*).

This 'cleaning up' would be because the country has indeed cleaned up its act as far as pesticide residues is concerned and now food is safe for consumption. But it could also well be because pesticide residue is now a serious public health concern and government laboratories would avoid 'finding' uncomfortable levels. It could also be that the laboratories are not 'looking' for the right pesticides. It is well known that farmers use newer varieties of pesticides and to find these in food, one needs tests to be designed for these pesticides. But

as the detailed analysis of the laboratories is unavailable in the public domain, it is difficult to know if the right tests are being conducted and if the right results are published. In this situation, it would be best to eat at our own risk.

14. *Is the government ensuring that pesticide usage is within the Acceptable Daily Intake (ADI) as directed by JPC?*

No. This is a crucial directive to ensure that our exposure to pesticides is not above the unsafe level (see Box: *Guidebook*).

But the FSSAI has still not set up its system to ensure that this information is collected and used. In fact, the work is preliminary and hesitant.

The Panel on Pesticide and Antibiotic Residues started to discuss this issue for the first and last time only in its fifth meeting held in October 2011. "It was observed that a lot of improvement is required to calculate the Theoretical Maximum Daily Intake (TMDI)."[13] The National Institute of Nutrition was requested to provide a database on age groups, body weight and food consumption patterns for different regions of the country.

15. *Why is measuring exposure to unsafe food so critical?*

Each country which uses and registers pesticides, must do its own homework and must ensure that exposure to each pesticide does not exceed the acceptable daily intake. This is the mandate of safety.

Therefore, we can use pesticides but only after ensuring that these toxins are kept within strict limits, which are not injurious to human health. Without this, the use of pesticides would be deadly. Indeed, it would be criminal.

But that said, we also know that India does not regulate its use of pesticides through this threshold of safety. What then is our situation? If we were to estimate our exposure to pesticides through our food, would it be within the acceptable daily intake?

16. *What did CSE do to calculate our exposure to pesticides?*

Step 1: We checked the ADI of key pesticides used in India. Besides JMPR, ADI is also fixed by the USEPA and the Australian government's Department of Health and Aging. Within them, the ADI varied for

DIETS OF THE WORLD: WHO/FAO REGIONAL DIETS

Commodity	Middle eastern	Far eastern	African	Latin American	European
Cereals	430.8	452.3	318.4	252.2	226.3
Root and tubers	61.8	108.5	321.3	159.3	242.0
Pulses	24.6	19.8	17.8	23.1	12.1
Total sugars and honey	95.8	50.5	42.7	104.3	107.3
Total nuts and oilseeds	12.8	50.0	34.2	57.5	29.9
Total vegetable oils and fats	40.3	14.2	23.3	21.8	36.6
Total stimulants	8.2	1.7	0.6	5.5	14.4
Total spices	2.5	3.0	1.8	0.5	0.5
Total vegetables	233.1	179.0	77.1	150.5	371.8
Total fish and seafood	13	34.7	36.5	45	46.3
Eggs	14.6	13.1	3.7	U.9	37.6
Total fruits	204.4	85.4	94.7	271.3	212.4
Milk and milk products	132.3	32.7	42.2	167.8	340.8
Meat and offals	71.3	47.0	30.4	78.0	217.3
Total animal oils and fats	1	1.9	0.7	5.5	10.7
Total diet in grams per person per day	1346.1	1093.8	1045.3	1354.1	1907.6

Note: All figures (in grams per person per day)
Source: World Health Organization

WHAT WE EAT: DAILY PER CAPITA INTAKE OF FOOD COMMODITIES IN INDIA

Product	Per capita supply (kg/year)	Per capita supply (gm/day)	Percentage of total diet
Total cereals	162	445	37.1
Total pulses	11	29	2.4
Total vegetables	87.32	239	19.9
Total spices	1.97	5.4	0.4
Total fruits	40.66	111	9.3
Total meat	5.2	14.2	1.2
Eggs	1.54	4	0.4
Fish	4.43	12	1.0
Milk, excluding butter	65.49	179	15.0
Total suger and honey	38.3	105.0	8.7
Animal fats (ghee, butter)	2.25	6	0.5
Vegetable oils	9.49	26	2.2
Oil crops	7.1	19	1.6
Treenuts	0.68	2	0.2
Total stimulants	0.74	2	0.2
Approimate average per capita daily diet		1200.0	

Source: Food and Agriculture Organization (FAO) 2001, Food Balance Sheet

many key pesticides. So CSE decided to estimate exposure twice — using the ADI of JMPR and then using the USEPA threshold.

Step 2: We needed data on what, and how much, Indians were eating. We decided to estimate the daily per capita consumption of various food commodities for India. For this, we used the FAO's Food Balance Sheet (FBS) 2001 data for India. The JMPR uses the same source to assess dietary intake. Also, this data is the most recent. The Indian government's published data on dietary patterns is valid only for the 1990s. The last dietary pattern data was published in 1998, giving the dietary pattern for early 1990s (see Tables: *Diets of the world* and *What we eat*).

Step 2A: CSE also wanted to check what the exposure was of society's most vulnerable group — small children. For this, CSE used the average diet of a 10 kg child (average of 1-3 years old, male and female child) from the India Nutrition Profile published by the Union ministry of human resource development in 1998. This was the most recent data available on the diet of children in India.

Step 3: For estimation of pesticide exposure, CSE used the theoretical maximum daily intakes (TMDIs) assessment model, prescribed by JMPR. This model is used as a screening methodology. In this model, pesticide exposure is calculated by multiplying established MRLs with the average daily consumption for each food commodity.

Exposure = MRL x diet

Using this formula, CSE calculated exposure to eight commonly used pesticides in India.

Step 4: CSE used the MRLs prescribed in the PFA, 1955 for the legal limits allowed in the food we eat.

17. *What did CSE find? How weak are Indian regulations that safeguard our health?*

We found our exposure is high and regulations, therefore, are meaningless. They do not adhere to any global norm as far as mandating safety is concerned.

What we found:

The theoretical maximum daily intake of various pesticides in India presents a horrific picture (see Table: *Monocrotophos mayhem*). The comparison of estimated daily intake of pesticides for a 60 kg adult

MONOCROTOPHOS MAYHEM

Estimating exposure to this pesticide

Food commodity	Indian MRL (mg/kg)	Diet (gm/day)	Pesticide intake (mg/day)	Distribution* (per cent)
FOR A 60 KG ADULT				
Wheat	0.025	158	0.0040	2.6
Rice	0.025	209	0.0052	3.5
Cereals, other	0.025	77	0.0019	1.3
Pulses	0	29	0.0000	0.0
Potatoes	0.05	43	0.0022	1.4
Tomatoes	0.2	20	0.0040	2.7
Onions	0.1	15	0.0015	1.0
Vegetables, other	0.2	160	0.0321	21.2
Condiments and spices	0	5	0.0000	0.0
Oranges and citrus fruits	0.2	20	0.0038	2.5
Other fruits	1	92	0.0921	61.0
Meat and poultry	0.02	14	0.0003	0.1
Eggs	0.02	4	0.0001	0.1
Milk	0.02	179	0.0036	2.4
Sugar and sweeteners[1]	0	105	0.0000	0.0
Animal fats	0.02	6	0.0001	0.1
Vegetable oil and crops[2]	0	45	0.0000	0.0
Tea, coffee and cocoa	0	2	0.0000	0.0
Total pesticide intake			0.1510	
ADI			0.0360	
Per cent of ADI			**419**	
FOR A 10 KG CHILD				
Cereals	0.025	119	0.0030	9.8
Leafy vegetables	0.2	7	0.0013	4.4
Roots and tubers	0.05	38	0.0019	6.3
Other vegetables	0.2	16	0.0032	10.6
Fruits[3]	0.86	20	0.0175	57.6
Meat, fish and eggs[4]	0.012	10	0.0000	0
Milk and milk products	0.02	164	0.0033	10.8
Pulses	0	20	0.0000	0
Sugar and sweeteners	0	19	0.0000	0
Oil and fats	0.02	7	0.0001	0.5
Condiments and spices	0	4	0.0000	0
Total pesticides intake			0.0305	
ADI			0.0060	
Per cent of ADI			**508**	

Notes: Monocrotophos JMPR ADI = 0.0006 mg/kg of body weight; *This means the proportion of pesticide intake through different food items [1] There is MRL for sugar beet, but none for sugarcane. Therefore, MRL has been assumed as 0. This is an under-estimation. [2] There is MRL for cotton seed oil but data for consumption is not available. Therefore, MRL has been taken as 0. This is an under-estimation. [3] Consumption data for fruits not available separately. MRL for fruits is weighted MRL of citrus fruits and other fruits using consumption pattern as per FAO 2001 FBS. [4] Separate consumption data not available. MRL for meat, fish and egg is weighted MRL, using consumption pattern as per FAO 2001 FBS. This is an under-estimation.

in India with JMPR's ADI shows that:

In adults...

Of the eight pesticides considered, TMDI for five exceeds JMPR fixed ADI for a 60 kg adult by 40 per cent to as much as 376 per cent. For DDT, the theoretical daily intake of a 60 kg adult in India is about 376 per cent higher; for monocroptophos, one of the most toxic pesticides widely used in the country, the theoretical daily intake of a 60 kg adult in India is about 300 per cent higher; for lindane, endosulphan and phorate, the theoretical daily intake is about 40 per cent, 95 per cent and 61 per cent higher, respectively.

But what is truly frightening is what we are doing to the young and vulnerable in our country. A 10 kg child — roughly three years old — is being literally poisoned each day through each meal.

In children...

Of the eight pesticides considered, TMDI for six exceeds JMPR fixed ADI by 14 per cent to a staggering 622 per cent.

Malathion and chlorpyrifos are the only two pesticides whose intake was within the 'safe' limit.

But wait. This is because the JMPR's assessment for these two pesticides allows extraordinarily high exposure. The ADI set by JMPR for these two commonly used pesticides greatly varies from the USEPA and Australian assessments. The JMPR ADI for malathion is as high as 18 mg/day for a 60 kg adult. JMPR had last reviewed malathion in 1997. The most recent review of malathion was done by USEPA in 2000 and based on this review, USEPA has put Chronic Reference Dose (CRfD), which is same as ADI, for malathion as 1.44 mg for a 60 kg adult.

Therefore, we decided to recheck. We ran the same model using, this time, the USEPA-set ADI.

What we found:

For 60 kg adults, of the eight pesticides considered, the theoretical maximum daily intake for seven exceeded the ADI by 49 per cent to 4,934 per cent. The theoretical daily intake of chlorpyrifos and malathion was now 1,309 per cent and 104 per cent higher... and for children it got really horrific.

Of the eight pesticides considered, the TMDI of seven pesticides exceeded USEPA fixed safety limits by 114 per cent to 7,118 per cent.

This level of pesticide intake through food (TMDI), allowed by the Indian law, as prescribed under the PFA Act, is truly disastrous. As a rule, globally, MRLs are set in a way that their dietary intakes do not exceed ADI. In India, it seems to be the opposite. Here, out of the eight most commonly used pesticides, the MRLs are set in such a way that the dietary intake for six of them exceeds ADIs as prescribed by JMPR. When compared with the USEPA's safety threshold, the dietary intake of seven out of the eight pesticides exceeds the levels. Therefore, the CSE's estimation of dietary intake of pesticides clearly shows that the current system of regulating pesticides in food commodities has failed miserably.

MEETA AHLAWAT / CSE

18. CSE's research was to determine the theoretical exposure of pesticides. What is the process through which the actual exposure can be assessed?

Most pro-active governments conduct what are called 'total diet studies' to estimate the actual exposure of pesticides and to reconfirm that pesticide exposure is within safety limits. In the US, the FDA conducts the total diet study to estimate people's actual exposure to pesticides through food. FDA buys food from supermarkets or grocery stores four times a year, once from each of the four geographical regions of the country. Four food baskets, comprising 261 food items, are prepared. These represent over 3,500 different foods reported in the US department of agriculture food consumption surveys (so, for instance, apple pie would represent all fruit pies and fruit pastries). The foods are cooked and then analysed for pesticide residues.

19. *What does this mean?*

This means that the food we eat is, by and large, unsafe for consumption. Or in other words, regulation is designed to ensure that food is unsafe for humans.

The standards that have been set — the legal limits that determine what is allowed in our food — are too high. These standards, even if enforced, which they are not, will not do very much for ensuring food safety. This is because the legal limit, if applied, will exceed the acceptable daily intake threshold of safety.

It is for this reason that we urgently need a complete revamp of the entire regulatory system so that human health is not compromised.

20. *So, should we harmonise our standards with Codex?*

No. That would be ridiculous. Government wants to harmonise Indian MRLs with MRLs set under Codex Alimentarius — the FAO-based global food standard agency. We need to harmonise our pesticide policy with our diets, not with Codex.

Once India regulates its pesticides based on its diet and ensures that it is within its acceptable daily intake advisory, then it can argue that diets cannot be harmonised and that its Indian standards have to be accepted as the standards for global trade as well.

21. *Should we then allow some pesticides in soft drinks and other processed foods?*

This would only be possible if we had space in our diet-pesticide calculation.

It is clear that living within the ADI quota requires a trade-off between poison and nutrition. What is 'safe' means calculating what we eat, how much we eat and how much pesticide can be allowed in all this. The food basket is also the pesticide basket.

Regulators have to ensure that there is 'space' in the ADI. That our theoretical maximum daily intake — what we are expected to eat in terms of nutrition and pesticides — does not exceed the ADI. But more importantly, that only a proportion of the ADI is used and that more and more accurate dietary-pesticide intake models allow governments to ensure that pesticides are not a health hazard because the exposure is much below ADI.

If exposure is much below ADI, governments can make adjustments for consumption of pesticides through other foods and media — like non-nutritive food or water or even air. You can ingest some 'unaccounted for' pesticides through non-essential food, which is not part of the daily diet-pesticide calculation.

Currently, we exceed the ADI because we have set very high MRLs. Because we exceed the ADI, we have no space for non-essential food. Remember, pesticide regulation is about the nutrition-poison trade-off. If our daily diet of pesticides is being exceeded only with essential food, we cannot allow pesticides in non-essential and non-nutritive foods. This is why we cannot allow pesticides in Coke or Pepsi.

But also, we will need to enforce the legal limits through an effective programme of surveillance and enforcement. We cannot argue that we cannot control pesticide contamination on our raw agricultural commodities and so cannot enforce standards for food safety. This is unacceptable and wrong.

Finally, we have to remember that regulation will cost. Every time, we register a pesticide for use we will have to incorporate this cost — of regulation and enforcement. Otherwise, we are discounting the real costs — of ill health and death. This is a deliberate policy of negligence. Criminal negligence.

22. *Has anyone in India calculated the actual exposure of pesticides?*

In 2009, the National Institute of Nutrition published the *Total Diet Study for Andhra Pradesh*[14] and the Directorate of Research, Bidhan Chandra Krishi Viswavidyalaya, based in Kalyani in West Bengal published the *Total Diet Study for West Bengal.*[15] The findings of Andhra Pradesh study are as follows:

1. The data on food consumption was taken from the Diet Survey Report 2006. In this study, commonly consumed foods were procured from the state of Andhra Pradesh and tested.
2. 258 samples of food and 24 samples of water were analysed for 19 pesticides. No sample was free from pesticide contamination.
   - Spinach had the highest concentration of cypermethrin in all vegetables; mango and milk followed.
   - Rice from Ramavaram mandal of East Godavari district had highest cypermethrin content.
   - Water had aldrin, chlorpyriphos and dieldrin.
3. Rice is the main staple diet and so exposure to pesticides through this was of concern.
4. Among the eight different physiological groups, children of 1-3 years and 4-6 years were more at risk from aldrin (4-6 per cent of the ADI) through exposure in rice and milk.
5. Although mango and green leaves were the most contaminated, these are consumed in small quantities and so exposure is limited.
6. In heavy metals, green vegetables and red chilli had the highest levels of cadmium.

23. *Does it mean that our exposure to pesticides is within safe levels?*

No. It only means that we do not know if our exposure is within safe levels. We do not know if the food we eat is, by and large, unsafe for consumption. This is because regulation is not designed to ensure that our food is safe.

24. *What is the agenda for safe food?*

Firstly, at the time of registration of pesticides, the ADI and the MRLs on all the food commodities should be established. The registration should not be done if the MRLs and ADI are not fixed. The MRLs should be established in such a way that the dietary intake of the pesticide does not exceed the ADI. ADI must become the cornerstone of all decisions taken regarding the use of pesticides.

Secondly, the enforcement of pesticide standards on food commodities needs a revamp so that data produced by laboratories is put in the public domain.

Thirdly, only less toxic pesticides should be registered. This process of registration, called 'comparative risk assessment' is becoming popular across the world and is a definite way to reduce the toxicity of pesticides used in the country. The country will have to invest in R&D to develop less toxic pesticides.

Fourthly, we must regulate pesticide use at the farm level. This we cannot do only by educating individual farmers, which so far the country has been trying to do unsuccessfully. To make it happen, we will have to regulate the supply chain of the pesticide companies.

Fifthly, pesticide residues in processed food commodities must be strictly regulated and minimised. To do this, we will have to develop MRLs for processed food commodities based on sound scientific and health-based principals and then implement them strictly. This is the only route left to us to reduce our pesticide intake.

25. *How do we break the circle of poison with new and safe chemicals?*

First, we must recognise that the more chemicals we register, higher is the cost of surveillance, residue analysis and enforcement. Can we afford this cost? Who will pay? In USA, managing pesticide risks cost 7.4 per cent of the gross pesticide sale between 1971-95. We cannot say that we are too poor to enforce health regulations once we have allowed use of a substance. Regulation has to be part of the use (registration) process. In the US, the pesticide industry is required by law to pay for the costs of re-registration. Indian pesticide industry must be asked for a cess to pay for enforcement of standards.

Secondly, we must note that in this business one toxic substance is replaced with another, even more lethal substance. This is exactly how global business works. So, if DDT is found to be noxious, companies find it much more profitable to 'substitute' it with an alternative. In 1972, DDT was found to be persistent. It was banned in the US. Industry then introduced alternatives: methoxychlor and dicofol, which were relatively close to DDT, and endosulfan. But as persistence was still a problem, a new pesticide-type was introduced: organophosphates. These pesticides have higher acute toxicity. They reduce the ability of the enzyme cholinesterase to regulate signals between neurons and can cause muscle weakness.

By the 1990s, concern for children's health grew in the industrial world. Scientists found that these new chemicals were less persistent but more toxic. Organophosphates were indicted for childhood developmental problems. By 2000, the US was asking for a review of organophosphates. It considered introducing a "common mechanism of toxicity", which will be able to better manage risks from cumulative toxic effects. It also planned to review 9,728 MRLs by August 2006.

It is important to break this toxic tango, which is deadly for our health and the environment. It is for this reason that we desperately need a global compact on product impact assessment and liability. The problem today is that the global ecological framework does not penalise the producer when their products are found dangerous or toxic. Instead, the global system actually rewards the producer by providing yet another lucrative market for its substitutes.

International agreements – such as the Stockholm Convention on Persistent Organic Pollutants and the Rotterdam Convention on Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade – do not address the principle of liability and compensation – or, in other words, holding the polluters liable to pay damages. Therefore, if inventors get incentives through the intellectual property system, the inventors whose products have adverse impacts should also stand to lose. There should be a global liability on each product.

This will force companies to do careful assessment and maybe create incentives for environment-friendly products.

In India, the agenda for safe use of pesticides is critical. But currently, the pesticide industry is on a warpath, targeting activists and scientists so that their voice is muzzled and research is compromised (see Box: *Pesticide industry targets activists*)

PESTICIDE INDUSTRY TARGETS ACTIVISTS

ANIL KUMAR / CSE

In the first week of April 2008, a group of men came and stood outside the CSE office. They carried placards with offensive slogans directed at me. We understood the 'protesters' were ostensibly from an NGO we believed was a front for the pesticide industry. We also understood the picket to be the latest in a dangerous pesticide industry mindgame.

Let me explain. For the past few years, the pesticide industry, represented by its rich and powerful owners, has held press conferences across the country slamming CSE's research on pesticide residues in food, in the blood of farmers in Punjab and in the soil, water and food of diseased and deformed villagers of Padre in Kerala. During this period, we have received dozens of legal notices from this industry, threatening dire consequences. Every time we have replied to these notices, stating the facts, there has never been a follow-up. Instead, another notice for some other frivolous reason gets sent. Initially, the industry targeted our research. The focus then moved to us – to CSE – before settling on me. A year ago, they hit a real low when they began circulating obscene cartoons of me that Rajju Shroff, owner of a leading pesticide company, had drawn.

In all this time, even as we refused to give in to the threats, we also respected their right to protest. This time, too, we decided to leave the picket alone. Then, a few days into the 'protest', a journalist with a city daily visited and recognised one of the protesters outside our gate. This was not an employee of the aggrieved pesticide company or a protesting NGO, he said. This man was a representative of a public relations company who had met him, on behalf of biscuit manufacturers, to make the case that government should allow processed food, instead of cooked hot meals, in the multi-crore school meal programme!

We were puzzled. Surely, Indian industry was too proud or forthright to hire protesters? Why would reputed public relations companies engage in dirty tricks and intimidation? We knew this kind of thing happened in the US, where corporations hired lobbyists and white collared goons. But was this now happening in India? We decided to investigate.

When we checked with all known names in the public relations business, nobody had heard of this company – Media Expressions Consortium. Finally, when my colleagues tracked it down to a small office based in a Mumbai suburb, a sinister canister of worms leaked out. The company, we learnt, represented the biggest of the polluters – the plastic industry and pesticide industry – as well as others, like the biscuit manufacturers, to defend their interests. The company boss proudly told my colleague he was out demonstrating in front of our office. But in the same breath he told her he had nothing to do with the protest. We realised why. His was a 'shadow' affair. This was the new face of Indian business – the hidden lobbyist who could skillfully make out cases for clients in different ways, from PowerPoint presentations to physical protest, all on hire, for a price.

Clearly this is now the toolkit of industry to deal with dissent – to suppress public opinion and to subvert decision-making via a fine public relations makeover. If you don't believe me just consider how, in this same period, the pesticide industry through its associations has filed countless cases against activists and scientists, but with an important difference. These cases derive from what is

known in the US as SLAPP – acronym for 'strategic lawsuits against public participation'. These are 'different' because the corporation (or its front organisation or lawyer) uses it not to get justice, but to threaten, intimidate and gag. The cases are filed not against institutions that can defend their interests but carefully target individuals and, in particular, professionals who refuse to prostitute their science to suit industry. The companies who file SLAPP cases rarely win in court, but make the defendants spend a huge amount of time and money running to the courts to fight the case. This harassment discourages others from petitioning government on public issues. Industry's business is served.

We first learnt about slapp when we released a study about pesticides in colas. PepsiCo had filed a defamation case against us in the Delhi High Court and our lawyer, fresh out of law school in Bangalore, jumped as he read through the company's petition saying this was a classic slapp case.

An environmental activist in West Virginia was sued for us $200,000 for criticising a coal-mining company for polluting the local river. Cattle-ranchers filed a million dollar case against television celebrity Oprah Winfrey for hosting a show on mad cow disease and discussing dangers of eating contaminated beef. The list runs in thousands.

The most (in)famous of these cases was filed by junk food giant McDonald's against two activists in Britain, who had in 1990 distributed a six-page leaflet on 'what was wrong with McDonald's'. The company accused them of defaming it because they had said that it contributes to cardiac diseases, cancer and diabetes. The company won the case in 1997 and it has become a precedent for corporate libel cases, commonly known as McLibel. Such cases particularly target individuals and media organisations so that the messenger is shot, along with the message.

Now we are catching up with the world.

Y S Mohana Kumar is a doctor practicing in a nondescript village called Padre in Kerala. Unknown, till he noticed that people in his village were more diseased and deformed than most and started asking questions. One thing led to another and researchers – from different institutions – confirmed and reconfirmed the presence of residues of endosulfan, a pesticide, in blood, soil and water samples from the village. In 2003, Mohana Kumar received a legal notice from the lawyers of the Pesticide Association of India threatening legal action if he did not apologise and withdraw his statements immediately. His crime? Writing a letter in this magazine on the findings against the government-appointed O P Dubey committee, which had absolved the pesticide of being the cause behind the deadly ailments of people in Padre.

For the record, Down To Earth followed up investigations against the Dubey committee and found to its horror evidence of how data was fudged; how scientists were coerced and how industry influenced the findings of the committee. The committee's proceedings were challenged and investigations reopened by the government. Mohana Kumar was right but that clearly was not the point.

Madhumita Dutta is not a doctor, but an environmental activist who recently received legal summons to appear before a court in Warangal, Andhra Pradesh. Her crime is that she researched and published, with others, an investigation on acute pesticide poisoning in the district. The case filed by the pesticide industry association Crop Care Federation even implicates the designer of the publication and is aimed at harassing and warning others to desist or be destroyed.

Umendra Dutt runs an ngo in Punjab called Kheti Virasat Mission, which works on various farmer-related issues, including pesticide use. He has been sued for Rs 5 crore by United Phosphorous Limited, a leading pesticide manufacturer. His crime: discussing in public, health studies on pesticide exposure and how it could act as a trigger to diseases, and evèn lead to congenital malformations and genetic disorders. All clearly well-established in scientific studies across the world.

But it does not stop there. The company has also filed a case against the media giant, Bennett and Coleman, the publishers of the Times of India. Their crime is similar: publishing a report quoting Dutt in their daily newspaper, Mumbai Mirror. The defamation case has been filed by the company alleging that the statements in the article will 'disparage our client's reputation' in the trade across the world. This is particularly intriguing, because the article does not mention the company at all, only pesticides and their health impacts.

But how do I know this? Because two weeks ago, my colleague Chandra Bhushan, received a letter from an ngo called the Centre for Environment and Agrochemicals, which enclosed a copy of this legal notice. The letter told him that if he was to attend a forthcoming meeting being organised by Kheti Virasat Mission he "will be made a party (to the case against Kheti Virasat) and unnecessarily dragged into litigation". In simple language, a simple threat: we will sue you if you dare to attend.

It does not stop there. We called to check more about the ngo and received another letter. The letterhead was the same, but the signatory had changed. Now Rajju Shroff, the owner of United Phosphorous Limited wrote, saying, "The industry has decided to take legal actions and expose all your activities." I am sure we will hear from them again.

In these modern David-and-Goliath tales, I can only hope (and pray) that there are many, many more Davids.

— Sunita Narain, *Down To Earth*, June 15, 2008

1.2 CHILDREN OF ENDOSULFAN

PHOTOGRAPHS: PUHANI KAUR / CSE

In April 2011, all 172 signatories (including India) to the Stockholm Convention on persistent organic pollutants agreed to phase out endosulfan in the next five years, extendable to a maximum of 10 years. The pesticide has already been banned in over 80 countries because of its harmful effects on human beings and ecology.

India sought an exemption for 14 crops for the 10 extendable years. However, in May 2011, the Supreme Court of India banned the production, sale, use and export of endosulfan. The ban came more than two decades after the demand for action gathered momentum in Kasaragod district of Kerala.

This ban on endosulfan is really a story of human misery, of dogged civil society activism, of use of science to push for better public policy, of industry's threats and intimidations, and of government complicity. CSE too played a role in this, as its Pollution Monitoring Lab was the first to test endosulfan poisoning cases in Padre village in Kasaragod.

1. What was the Supreme Court judgement on endosulfan?

On May 13, 2011, the Supreme Court banned the manufacture, sale, use and export of endosulfan throughout India, citing its harmful health effects. This landmark judgment came on the heels of India's grudging acceptance at the Stockholm Convention that endosulfan is a serious health hazard and that it should be banned (see Box: *Banning the killer*). The three-judge bench, comprising chief justice S H Kapadia, justice Swatantar Kumar and justice K S P Radhakrishnan passed the order in response to a hearing of a public interest litigation filed by the Democratic Youth Federation of India (DYFI-Kerala wing) seeking a ban on the toxic pesticide.

The ban was to remain effective till the time the joint committee, formed under the aegis of the Indian Council of Medical Research (ICMR) and the agriculture commissioner, submitted its report to the court about the harmful effects of the widely used pesticide. The report was expected to be filed within eight weeks. The committee, however, filed a sketchy report stating there had been serious health impacts due to the unscientific use of endosulfan in Kerala and Karnataka. It also agreed that there are no studies done by states to show the impacts of endosulfan.

BANNING THE KILLER

- **On the final day of the Conference of Parties (CoP) 5 to the Stockholm Convention – April 29, 2011 – endosulfan was brought under Annex A of the Convention, which meant that it was to be banned globally; 80 countries had already banned endosulfan, and at CoP 5 all the 172 participating countries agreed to the inclusion of the pesticide in Annex A.**
- **Endosulfan has been listed under Annex A with exemptions, meaning a global ban on all production and use within five years. This five-year period can be extended to a maximum of 10 years. After that, it will take another one year for the ban to be executed. India, China and Uganda are the three countries that have asked for exemptions for certain pests for cotton, coffee, tea, jute, apple, tobacco, cow peas, beans, tomatoes, okra, eggplant, onion, potatoes, chillies, mango, gram, arhar, maize, rice, wheat, groundnuts and mustard. For the rest of the countries that are not asking for exemptions, the ban takes effect within one year.**
- **In India, the Supreme Court ordered a ban on May 13, 2011 on the sale, use, production and export of endosulfan.**

Apart from conducting a pan-India study on the health impacts of endosulfan, the court has also ordered the government to come up with safer and cheaper alternatives of the pesticide and decide on how existing stocks can be disposed.

Soon after the ban was announced, Hindustan Insecticide Limited's (HIL) license to produce endosulfan was cancelled by the Kerala government on May 27, 2011. The Gujarat agriculture department seized the permits of nearly 80 endosulfan manufacturing and formulating units in the state. The production and sale of endosulfan from these units has stopped. Similarly, endosulfan units in Maharashtra had been issued orders to stop manufacture and sale of the pesticide.

The Supreme Court, on September 30, 2011 allowed exports of 1090.596 metric tonne (MT) of unused endosulfan for which export orders had been received. But it reiterated its earlier order, banning the use, sale and production of the pesticide.

The ICMR-agriculture commissioner joint committee justified exports by stating that the industry had no experience of disposing of the pesticide and that the ministry of environment and forests had not provided any definite procedures for the purpose.

On December 13, 2011, the Supreme Court also allowed the export of raw endosulfan for which the export orders had not yet been received. This order made it clear that the court was definitely not in favor of endosulfan being used within India.

2. *Why did the Court ban endosulfan?*

The Supreme Court decided to ban this pesticide on account of its adverse health and ecological impacts (see Box: *Endosulfan and its health impacts*). The court took into account the horrendous fallouts the use of this pesticide has led to in Padre village in Kasaragod district of Kerala.

3. *What is the endosulfan case in Kerala all about?*

Padre is a village in Enmakaje panchayat in Kasaragod district. This village has been the center-point of endosulfan controversies for more than two decades (see Box: *Timeline endosulfan*).

Around 1963-64, the Kerala agriculture department began planting cashew trees on the hills around Padre. The Plantation Corporation of Kerala (PCK) took over the cashew estate in 1978.

A pesticide called endrin was sprayed to counter the tea mosquito, a major pest that affects the yield of cashew. Later, PCK began spraying endosulfan. Aerial spraying of endosulfan began around 1976, though the PCK maintained that aerial spraying was initiated only in the 80s. From 1981, PCK started regular spraying of endosulfan two to three times a year. The *Evidence Weekly* magazine published a report (December 25, 1981) on cows giving birth to calves with deformed limbs after aerial sprays of endosulfan in Enmakaje gram panchayat. The author was Srikrishna 'Shree' Padre, a farmer and journalist covering agriculture, environment and matters of public interest.

Soon, protests against aerial spraying began. The panchayat complained to the district collector, seeking a thorough probe. Residents saw honeybees die, and frogs and fish disappear from the area. Farmers producing honey to supplement their incomes opposed the aerial spraying.

Reports of unusual diseases among villagers in Padre began trickling in from the end of the 1990s. Mohana Kumar Y S, a doctor who has been practising medicine in the area since 1982, started recording disorders of the central nervous system among the children of the area — cerebral palsy, retardation of mental and/or physical growth, epilepsy and congenital anomalies like stag horn limbs. There

ENDOSULFAN AND ITS HEALTH IMPACTS

Endosulfan is an organochlorine insecticide and acaricide, and acts as a contact poison on a wide variety of insects and mites. It is widely used on cereals, coffee, cotton, fruit, oilseeds, potato, tea, vegetable and other crops. It is also be used as a wood preservative. Commercially produced endosulfan usually consists of its two molecular forms (isomers) a-endosulfan and b-endosulfan.

There is a global concern about the acute and chronic toxicity of endosulfan. The pesticide is a known carcinogen, neurotoxin and genotoxin (damages DNA); the Insecticides Act of 1968 recommends its restricted use.

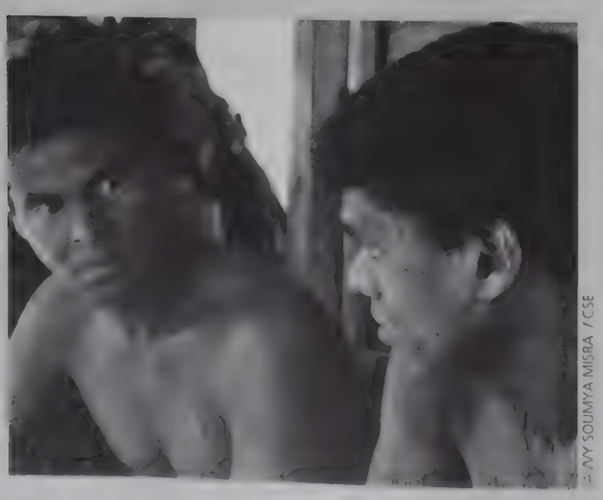
Endosulfan is easily absorbed by the stomach and lungs and through the skin, which means all routes of exposure can pose a hazard. Exposure to endosulfan may result from breathing the air near where it has been sprayed; drinking water contaminated with it; eating contaminated food; touching contaminated soil; smoking cigarettes made from tobacco with endosulfan residues; or working in an industry where endosulfan is used. Proper protective clothing (safety goggles, gloves, long sleeves, long pants, respirator) is needed to prevent poisoning when handling endosulfan.

Endosulfan has been shown to have higher acute oral and inhalation toxicity. Acute endosulfan poisoning can cause convulsions, psychiatric disturbances, epilepsy, paralysis, brain oedema, impaired memory and death. Its toxicity is so high that it can be fatal if inhaled, swallowed or absorbed through the skin in high amounts. Exposure through certain conditions of use (such as lack of protective equipment) and 'bystander'exposure have been linked to congenital physical disorders, mental retardation and death in farm workers and villagers in developing countries.

Endosulfan persists in the environment and bioaccumulates in animals and plants, leading to food contamination and eventually, dietary exposure in humans. Its predominant toxicological effect is over-stimulation of the central nervous system. Long-term exposure is linked to immuno-suppression, neurological disorders, congenital birth defects, chromosomal abnormalities, mental retardation, impaired learning and memory loss. There is some indication that endosulfan can have adverse effects on the immune system at low levels of exposure. The immune system is adversely affected because exposure decreases the white blood cell count.

There are several studies that prove that endosulfan is an endocrine disruptant. A relationship has been observed between maternal exposure and foetal malformations; physical malformartions observed in humans include cleft palates, harelips, club feet, limb malformations, eye deformities and extra fingers and toes.

Endosulfan is also known to have severe ecological impacts. It is toxic for bird species, fish and aquatic species and bees.



JAY SOUMYA MISRA / CSE

were too many cases of cancer of the liver and blood; infertility and undescended testes among men; miscarriages and hormonal irregularities among women; skin disorders; and asthma, to name a few. Psychiatric problems and suicidal tendencies were also rising. Surprisingly, almost all the ailments were restricted to people less than 25 years of age. As no other visible sources of pollution existed in these areas, the diseases left everyone perplexed; some blamed it on a supernatural curse.

From 1997 onwards, social groups became active in the district highlighting the plight of the people in Padre. The matter came to a head on December 25, 2000, when PCK announced that it would carry out aerial spraying the next day. The people of Padre gathered around the temporary helipad to protest and asked PCK officials to desist from aerial spraying. When the crowd became agitated, PCK called in the police and aerial spraying was carried out under police protection.

The residents of the area came together and formed the Endosulfan Spray Protest Action Committee (ESPAC). The committee also involved Shree Padre and Mohana Kumar, and began discussing the linkages between endosulfan and the unusual ailments in the surrounding villages. It wrote letters to the Kerala government and to the Kerala Agriculture University (KAU), demanding a withdrawal of the certification of the pesticide. The KAU set up a committee to conduct a study on 'Environmental effects of aerial spraying on cashew plantations in Kasaragod'. Meanwhile, civil society groups from Kerala approached CSE in late 2000 to conduct a study in Padre village and determine the health impacts caused due to aerial spraying of endosulfan in the area (see next question).

The endosulfan menace has spread. People in nearly 11 more panchayats in Kasaragod are now affected. Victims are suffering from congenital deformities, physical disabilities, mental retardation and gynecological problems. The same health impacts are now being seen in the neighbouring Dakshin Kannada district in Karnataka. Here, the Karnataka Cashew Development Corporation aerially sprayed endosulfan over cashew plantations for over 20 years.

4. *What is the CSE study on endosulfan in Padre village?*

In 2001, the Pollution Monitoring Lab (PML) of CSE collected and analysed 25 samples from Padre for testing endosulfan residues; these samples were collected a few days after the aerial spray. The samples included soil from the village and from the plantation; surface water from the local streams and tanks which are drinking water sources; samples of cow's milk which were fed with fodder from in and around the plantation; blood sample of villagers; milk sample from a lactating mother; samples of fish, vegetables, coconut oil, butter and spices; cashew nut and cashew leaves and a live frog.

The samples were analysed with gas chromatography technique with electron capture detector. The USEPA methodology was used.

5. *What were the findings of the CSE study?*

The study found that extremely high levels of endosulfan were present in all the samples.

The CSE report, released on February 21, 2001, concluded: "Alarmingly high values of endosulfan residues (ppm) for blood, fruits, tissues only go to prove the high diseased condition in the people of Kerala. It is beyond doubt that the dramatic cases of endosulfan poisoning in Padre village in Kasaragod district of Kerala (PCK plantations) can be directly linked to a decision-making process dominated by a government undertaking which has resorted to aerial spraying of endosulfan thrice a year for the last 25 years, without sufficient back-up from, or debate with, experts in other disciplines, including pesticide experts, social scientists, environmentalists and others (see Table: *The findings*)."

6. *What other studies were done after the CSE study and what were their findings?*

The CSE study triggered numerous studies and probes by the state and Central governments as well as by the civil society. Agencies like the ICMR visited the area, and conducted studies and surveys to understand the relation between aerial spraying of endosulfan and the health problems in the village. The results were mixed. A few studies acknowledged that ecological and health hazards in Padre were due to endosulfan poisoning; some rejected the cause-and-effect relationship. Yet others remained neutral, demanding more studies. But all studies recommended banning of aerial spraying of endosulfan in the area, accepted the evident lapses in precautionary measures by PCK, and acknowledged the fact that there were indeed a large number of people with health abnormalities. The findings of some of the important studies/probes are as follows:

A. Achyuthan Committee Report, 2001

Reacting to the CSE report, the government of Kerala issued an order on February 23, 2001 to the state agriculture department to appoint a committee to study the endosulfan problem and its effects on humans and environment. A Achyuthan, an environmentalist based in Kerala, was asked to chair

THE FINDINGS OF THE CSE STUDY

Sample	Detected value of endosulfan	Maximum residue limit (MRL)	Number of times value exceeds MRL	Site/source of sample
Water	1.18	0.18*	7	Small stream in Kumbdaje village near Padre
Water	6.87	0.18*	38	Tank near the cashew plantation, Kajampady, Padre
Water	9.19	0.18*	51	The Kodenkiri stream near Vaninagar, Padre
Butter	14.00	NA	NA	From the milk of a cow of Saletadka, Padre
Cow's skin/fat tissue	49.99	0.1	500	From the abdominal region of a cow from Padre
Cow's milk	31.80	0.5	64	From a cow that grazes around Kajampady, Padre
Cow's milk	57.20	0.5	114	From a stall-fed cow in Kumbdaje village
Coconut oil	17.00	NA	NA	Extracted from produce of trees in Vaninagar, Padre
Cashew	54.11	NA	NA	From a tree in the plantation near Kumbdaje village
Vegetables	31.24	0.4-2.0	78-16	Basale; leafy, spinach-like vegetable, from Kajampady
Human milk	22.40	NA	NA	Lalitha, 35, resident of Kumbdaje village
Human blood	108.90	NA	NA	Vishnu Kulkarni, 16; has epilepsy & mental retardation
Human blood	114.13	NA	NA	Prabhawati Shastri, 35; has asthma & skin allergies
Human blood	115.19	NA	NA	Mohana Kumar, 40; has chronic throat infection
Human blood	109.50	NA	NA	Kittanna Shetty, 21; has cerebral palsy
Human blood	196.47	NA	NA	Muthakka Shetty, 50; Kittanna Shetty's mother
Human blood	176.90	NA	NA	Lalitha, 35, resident of Kumbdaje village
Live frog	10.35	NA	NA	From a small stream in Kumbdaje village
Cashew	3.74	NA	NA	From the plantation near Kajampady, Padre
Spices	212.28	NA	NA	Pepper bunch from Kajampady, Padre
Fish	28.24	NA	NA	From a tank in Kajampady, Padre
Soil	35.16	0.09*	391	From Lalitha's house in Kumbdaje village
Soil	3.17	0.09*	35	From a few metres inside the plantation at Kajampady
Soil	6.40	0.09*	71	From plantation area on a hilltop in Periyal, Padre
Cashew leaves	6.52	NA	NA	From the heart of the plantation at Periyal, Padre

Notes: All figures are in parts per million (ppm). 1: Values are the sum of a-endosulfan and b-endosulfan residues. Levels of endosulfan sulphate were not measured. Had this been done, the figures would have been higher. 2: The MRLs are for the sum total of a-endosulfan, b-endosulfan and endosulfan sulphate residues. Calculated from documents of the US Environmental Protection Agency. *: The MRLs for water and soil are the sum total of a-endosulfan and b-endosulfan residues, and do not include the MRL for endosulfan sulphate. NA: MRLs not available

the committee.

On November 22, 2001, the committee released its report. It did not collect samples; instead, it examined data collected by other agencies and recorded written and oral depositions from concerned authorities.

The committee suggested that though there is no direct evidence to attribute health problems to endosulfan, there was no evidence to completely deny it either. The committee concluded that the undulating nature of land and presence of a large number of water bodies and human habitation in and near the plantations made the area unsuitable for aerial spraying of pesticides; the protocol for aerial spraying was not followed by PCK either. The study pointed out that the use of the same pesticide for 20 years was against the recommended practice of rotating pesticides. The committee recommended banning of aerial spraying of pesticides in all cashew plantations of PCK in Kasaragod and that the use of endosulfan should be frozen for five years in these plantations. Further, it said that plantations in Perla division (which included Enmakaje panchayat) should observe five years of pesticide holiday.

B. Kerala Agriculture University Study, 2001

The preliminary report by the committee constituted by the Kerala Agriculture University (KAU), headed by M Abdul Salam, associate dean of the college of agriculture, was released in February 2001. The committee, set up on February 13, 2001, was to conduct a study on 'Environmental effects of aerial spraying on cashew plantations in Kasaragod'. This committee was constituted based on a letter that ESPAC had written to KAU in January 2001, demanding a clarification on why the spraying of endosulfan was recommended for cashew plantations, as well as calling for the withdrawal of certification of the pesticide.

The KAU was supposed to make its preliminary report on the basis of visits to the village on February 19, 2001 (50 days after the last spray). Subsequent reports were to be filed after visits in August and a brainstorming session on the use of endosulfan. Preliminary reports on 15 water and soil samples showed that though no endosulfan residues were detected in water, residues were high in the soil inside the plantation and low in the soil from adjoining areas. The KAU team recommended an immediate stop to aerial spraying and a need-based application of the insecticide.

The second study also recommended a stop to aerial spraying. It acknowledged the unusual human health problems in Padre village, but did not establish a cause-effect relationship between endosulfan and these problems. The study said that aerial spraying in the state should be stopped as the safety measures were inadequate. It also recommended in-depth health studies along with studies on water pollution, heavy metal contamination and natural radioactivity. The report concluded that there was no clear evidence to prove that endosulfan was the cause of the problem. ESPAC contested this report.

C. Frederick Institute of Plant Protection and Toxicology (FIPPAT) Report, 2001

FIPPAT, a Tamil Nadu-based private laboratory, also known as the International Institute of Biotechnology and Toxicology, did a report on pesticide residues in Padre. The study was sponsored by PCK. It collected samples two months after the last spraying and residues of endosulfan were analysed in 106 samples of human blood, along with cow's milk, fish, water, soil and cashew leaf samples collected from Padre. FIPPAT results showed no residues of endosulfan in any of the blood samples, cow's milk and water samples. However, some residue was detected in the soil (0.001 to 0.012 ppm) and leaf (0.04 to 2.863 ppm) samples. The pesticide industry used this report to rubbish the CSE study. However, when CSE analysed the full report it found that FIPPAT had actually detected endosulfan residues in human blood samples but had not disclosed this in its final report.

D. National Institute of Occupational Health Report, 2002

India Today magazine published a report 'Spray of Misery' in July 2003, documenting the physical and mental illnesses faced by people due to endosulfan. The National Human Rights Commission (NHRC) took *suo moto* cognisance of the report and asked the Indian Council of Medical Research (ICMR) for a detailed study. A three-member team from the National Institute of Occupational Health (NIOH) was set up to do this study. The NIOH report was released

THE GOVERNMENT SUPPRESSED FACTS

A Right to Information application filed by C Nithyanand Pai, convener of the Consumer Welfare Forum in Puttur (Karnataka), shows that the Indian Council of Medical Research (ICMR) had funded a project titled 'Effect of long-term endosulfan exposure on male fertility' in 2003, but never released the study despite it being completed in 2006. A team of doctors from the Kasturba Medical College, Manipal, conducted the study.

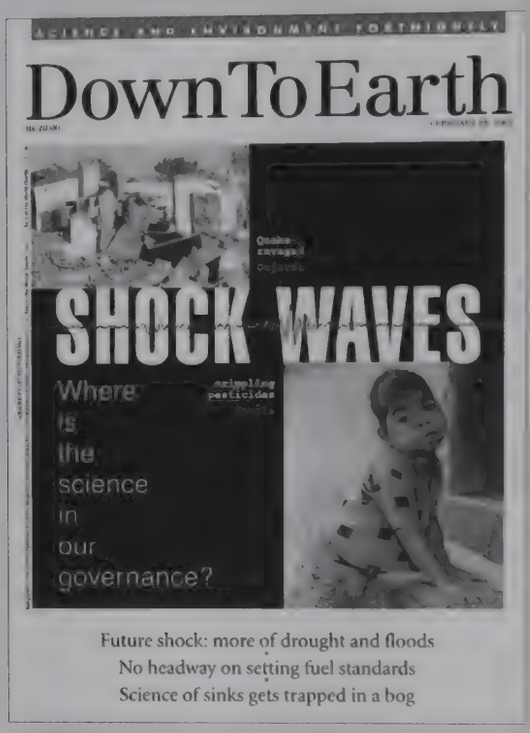
The study concluded that reproductive hormones like follicle stimulating hormones (FSH) and testosterone levels were significantly reduced in the endosulfan-exposed population compared to those who were not exposed. The cytogenetic study also revealed significantly higher amounts of genetic damage compared to unexposed control objects.

Another report published by the Kerala State Council for Science, Technology and Environment (KSCSTE) concludes that endosulfan is still persistent in the soil and sediment samples in Kasaragod. The study was conducted in 11 affected panchayats in the district.

As per this report, soil samples from Bellur (25.96 ppb) had the highest residues. Muliyar (19.69 ppb) panchayat had the highest endosulfan residues in sediment samples collected from ponds and valley slopes. The study also stated that nearly 40-70 per cent of plant diversity declined; honeybee populations were also affected.

TIMELINE ENDOSULFAN

- February 21, 2001: CSE releases its pesticide monitoring study report on Kasaragod, which documents the health issues in the area.
- February 23, 2001: Kerala government issues an order to the state agriculture department to appoint a committee to study the endosulfan problem and its effects on humans and environment. The committee is chaired by A Achyuthan.
- February 28, 2001: Preliminary report by the committee constituted by the Kerala Agriculture University (KAU) is submitted. KAU team recommends an immediate stop to aerial spraying and need-based application of insecticide.
- June 4, 2001: Frederick Institute of Plant Protection and Toxicology (FIPPAT) releases its report on pesticide residue analysis in Padre. Results show no residues of endosulfan in any of the blood samples, cow's milk and water samples.
- July 23, 2001: Indian Council of Medical Research (ICMR) sets up a three-member team from the National Institute of Occupational Health (NIOH).
- August 25, 2001: Government of Kerala suspends the use of endosulfan in all crops and plantations until further orders.
- November 22, 2001: Achyuthan committee releases the 'Report of the committee to study and analyse the effects of aerial spray of endosulfan in the cashew plantations of PCK in Kasaragod district'.
- January 2002: Three-member NIOH team releases the first part of the study. Report concludes that there is a high prevalence of congenital malformations in the exposed groups, low IQ, scholastic backwardness, learning disability, early menarche in girls and delayed puberty in boys.
- February 2002: Thanal, a non-profit in Kerala, releases its report 'Long-Term Monitoring – The impact of pesticides on the people and the ecosystem in Kasaragod, Keralam, India'. The survey by Thanal is on various types of disorders prevalent in the district and the number of people suffering from them.
- February 18, 2002: Government of Kerala issues orders on continued prohibition of aerial spraying of endosulfan on all crops in the state (see August 25, 2001), five-year pesticide holiday for Perla division and only ground-based spraying (manual spraying) to be undertaken by PCK in cashew plantations.
- February 20, 2002: Inter-ministerial committee at the Centre discusses the NIOH report. Expert committee chaired by Dubey set up to examine the reports of NIOH, Achyut Committee, KAU and FIPPAT.
- July 15, 2002: Connivance of the pesticide industry v government officials and scientists in Kerala Agricul University in an effort to get a clean chit for its produ exposed by Down To Earth.
- July 24, 2002: NIOH's second report finds higher prevale of neurobehavioural disorder and congenital malformati in females and abnormalities in male reproductive syste Enmakaje panchayat as compared to the control grou Meenja panchayat.
- August 12, 2002: Kerala H Court bans use of the endosu in the state pending a decis from Dubey committee. Inte order is passed on two PILs f by the Thiruvamkulam Nat Lovers Movement, the Peop Council for Social Justice and Samatha Law Society, seekin ban on the deadly pesticide.
- September 4, 2002: The Hea and Family Welfare Departm of Kerala forms an exp committee under chairmanship of P K Sivaraman look into the health hazards endosulfan in the area under I and do a detailed study.
- April 1, 2003: O P Dul committee report is discussed the 233rd meeting of Registration Committee. Dubey committee report establishes no links between use endosulfan in PCK plantations and health problems repor in the Padre.
- August 2003: P K Sivaraman committee submits its repo recommends a permanent ban on aerial spraying, a enhancement of medical facilities.
- April 15, 2004: Down To Earth reveals how the Dul committee was manipulated, evidence was suppressed a facts were distorted to absolve endosulfan.
- September 2004: The government forms another exp committee under C D Mayee, agriculture commissioner. T committee is asked to examine the various reports, findi and recommendations of previous committees endosulfan in order to assess its safety in regard to pul health and to make recommendations regarding the fut use of endosulfan.
- December 2004: Mayee committee submits its report. T report (marked 'For official use only') neither re-examin



Future shock: more of drought and floods
No headway on setting fuel standards
Science of sinks gets trapped in a bog

the FIPPAT issue nor does it relook into matters that the Dubey committee had ignored. Mayee gives a clean chit to endosulfan, says no links between endosulfan spray and health problems.

December 7, 2004: State Pollution Control Board suspends use of endosulfan in any form in Kasaragod until a final decision is taken in the matter.

December 13, 2005: Union ministry of agriculture issues gazette notification restricting the use of endosulfan in any form in Kerala.

June 3, 2006: Centre for Environment & Agrochemicals (CEA) sends notice to NIOH asking for unconditional apology in writing and for withdrawal of the NIOH report with immediate effect. CEA says the study is unfounded and unscientific.

May 2-3, 2007: CEA hires a PR agency to distribute booklets called 'Chemistry of a scientific fraud', near the CSE office; booklet contains obscene cartoons which target Sunita Narain, CSE's director general. The agency also pickets the CSE office for about a month.

October 13-17, 2008: Endosulfan enters the process of consideration as a Persistent Organic Pollutant (POP) in the fourth meeting of the Persistent Organic Pollutants Review Committee (POPRC) of Stockholm Convention.

October 12-16, 2009: Fifth meeting of POPRC reviews and adopts a revised draft risk profile on endosulfan, according to which it agrees that the POP characteristics of the chemical warrant global action.

March 15-19, 2010: India blocks the listing of endosulfan in Annex III of the Rotterdam Convention at the Chemical Review Committee meeting. Under Annex III, a chemical is subjected to Prior Informed Consent (PIC) of the importing country.

June 2010: US bans endosulfan.

October 11-15, 2010: Sixth meeting of POPRC agrees to adopt the risk management evaluation for endosulfan and recommends listing endosulfan in Annex A of the Convention, a move that can lead to its elimination from the global market. India opposes the move.

November 18, 2010: National Human Rights Commission issues notices to the Central and state governments seeking explanations on reports that aerial spraying of endosulfan in Kasaragod had affected the people severely.

November 19, 2010: Kerala Pollution Control Board issues a notification to ban use of endosulfan in the state under Water (Prevention and Control of Pollution) Act, 1974 and Air (Prevention and Control of Pollution) Act, 1981.

February 17, 2011: Karnataka bans endosulfan.

April 25-30, 2011: CoP 5 of the Stockholm Convention is held at Geneva. India agrees to the listing of endosulfan and its related isomers in Annex A of the Convention, without any opposition but with exemptions. Listing of endosulfan in Annex A means it should be banned globally.

May 13, 2011: Supreme Court bans the use, sale, production and export of endosulfan across the country. The Court passes the order while hearing a petition filed by the Democratic Youth Federation of India, and sets a two-month deadline for the ICMR-agriculture commissioner joint committee to submit its report on the health impacts of endosulfan.

in two parts in January and July 2002.

The first report, released in January, showed presence of endosulfan residues in samples of water as well as blood from Padre. The report concluded that there was a high prevalence of congenital malformations in exposed groups, low IQ, scholastic backwardness, learning disability, early menarche in girls and delayed puberty in boys.

The second report, released in July, found higher prevalence of neuro-behavioral disorder and congenital malformations in females and abnormalities in male reproductive system in Enmakaje panchayat as compared to control group in Meenja panchayat. The study concluded that the health problems in the Enmakaje panchayat were due to the high and continued exposure to endosulfan through various environmental media such as food, water, soil and air. It also added that there was a close similarity between the spectrum of health effects observed in the study population and those described in animal experiments. The NIOH study indicted endosulfan.

E. O P Dubey Committee Report, 2002

After NIOH submitted its report, an inter-ministerial committee was set up at the Centre to discuss the NIOH report. In February 2002, this committee sought the view of the Insecticides Registration Committee (constituted under the Insecticides Act, 1968) on the endosulfan issue. The Registration Committee in turn constituted an expert group chaired by O P Dubey, assistant director general (plant protection) of the Indian Council of Agricultural Research (ICAR), New Delhi. The Dubey Committee was to examine the reports of the NIOH, the Achyuthan Committee, KAU and FIPPAT and do a safety assessment of endosulfan, with the aim of recommending its continued or restricted use or otherwise.

The committee's report, released in March 2003, established no links between use of endosulfan in PCK plantations and health problems reported in Padre village. It recommended a comprehensive and detailed health and epidemiological study in the entire cashew plantation area of Kerala in order to establish a relationship between illnesses in Padre village and endosulfan. The government of Kerala was asked to conduct this study. The committee said that in view of the recommendations of the other committees, aerial spraying of pesticides may not be allowed at all in any situation.

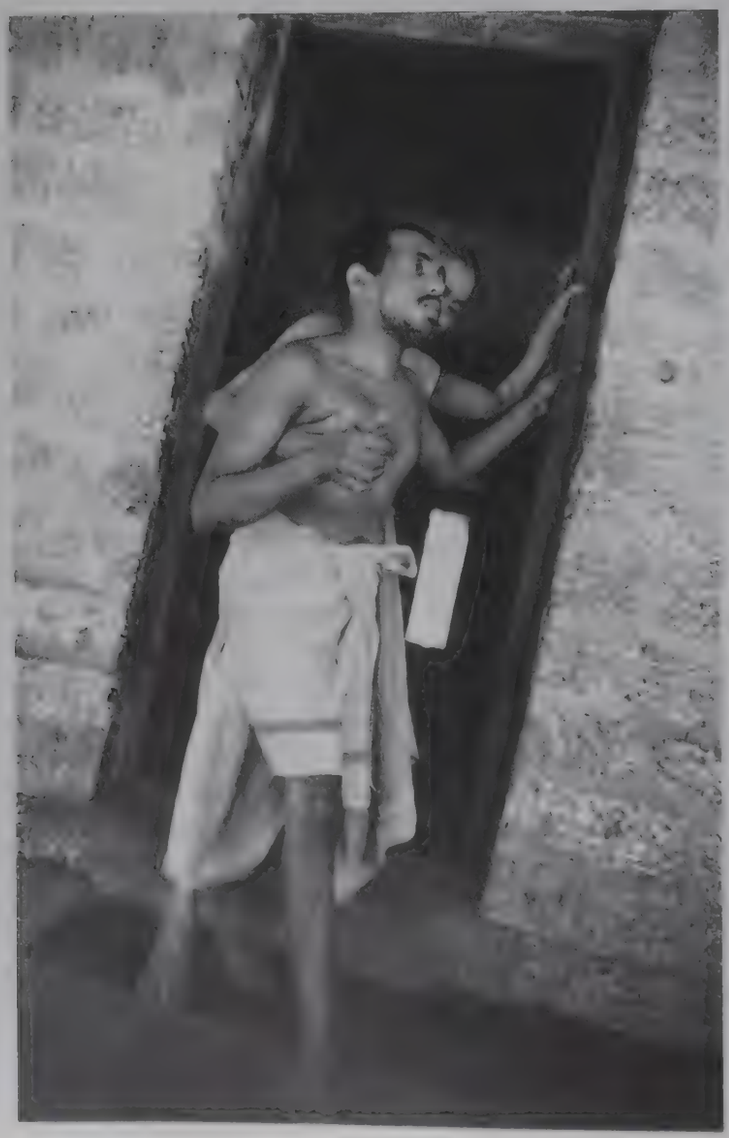
CSE's investigations revealed that the O P Dubey committee was manipulated, evidence was suppressed and facts were distorted to absolve endosulfan. The committee completely ignored the

NIOH report or the answers given by NIOH to the queries posed by the Dubey committee itself. Instead, it relied completely on the industry-sponsored FIPPAT report.

F. P K Sivaraman Committee Report, 2003

The Health and Family Welfare Department of Kerala formed an expert committee under the chairmanship of P K Sivaraman, additional director of health services at the directorate of health services, Kerala. The committee was constituted to look into the health hazards of endosulfan in the area under PCK and do a detailed study.

The committee submitted its report in August 2003. It concluded that high levels of endosulfan residues in the blood samples of school children in and around the PCK plantations showed long term exposure to the pesticide. It also concluded that PCK did not follow protective precautions for the workers, the public in the surrounding areas and the water sources during spraying. Since there was no other source that could explain the health hazards, the committee attributed it to aerial spraying of endosulfan in the plantation.



The committee recommended a permanent ban on the aerial spraying, and enhancement of medical facilities, especially in the affected area of Kasaragod. It also recommended regular monitoring of the people for similar anomalies and fresh cases in the affected area by health workers, and regular reporting to the district medical officer.

G. Mayee Committee Report, 2004

Due to lack of lack of unanimity among the members of the Dubey committee, in September 2004, the Central government formed another expert committee under the chairmanship of C D Mayee, agriculture commissioner. The Mayee committee was asked to examine the various reports, findings and recommendations of previous committees on endosulfan in order to assess the safety with regard to public health and to make recommendations regarding the future use of endosulfan. The committee gave a clean chit to endosulfan in its report, which was marked 'For official use only'. It did not investigate some of the key slippages by earlier committees – such as the issue of suppression of facts by FIPPAT, or the way the Dubey committee ignored various essential factors.

7. What actions were taken by the state and the Central governments in response to the various studies and findings?

- **On August 25, 2001,** the government of Kerala issued orders to suspend the use of endosulfan for all crops and plantations until further notice.
- **On February 18, 2002,** the Kerala government modified its previous order and issued another order specifying prohibition of aerial spray of endosulfan on all crops in the state, a five-years pesticide holiday for Perla division (where Padre is located), and only ground-based spraying (manual spraying) to be undertaken by PCK in the cashew plantation.

This was while the Pesticide Manufacturers and Formulators Association of India (PMFAI) was moving the Kerala High Court, seeking to quash the state government order of August 25, 2001 (to suspend the use of endosulfan in the state); the High Court bench decided that the government should take cognisance of the Achyuthan Committee report and take a decision based on Section 27(1) of the Insecticides Act.

According to this Section, If on receipt of a report, the Central government or a state government is of the opinion that the use of any

insecticide is likely to involve such risk to human beings or animals as to render it expedient or necessary to take immediate action, then that government may, by notification in the official gazette, prohibit the sale, distribution or use of the insecticide or batch, in such area, to such extent and for such period (not exceeding 60 days) as may be specified in the notification pending investigation into the matter.

- **On August 12, 2002,** the Kerala High court banned the use of endosulfan completely in the state pending a decision from the Dubey Committee. The division bench made it clear that the pesticide cannot be used in any of its formulations or under any of its brand names. The interim order was passed on two PILs filed by the Thiruvamkulam Nature Lovers Movement, the People's Council for Social Justice and the Samatha Law Society that sought a ban on the deadly pesticide.
- **On December 7, 2004,** the Kerala State Pollution Control Board suspended the use of endosulfan in any form in Kasaragod district until a final decision was taken in the matter.
- **On December 13, 2005,** the Union ministry of agriculture issued a gazette notification restricting the use of endosulfan in any form in Kerala.

8. *What has been the pesticide industry's reaction to this entire episode?*

Pesticide manufacturers have tried all possible tricks in the book to to deny and reject any findings linking endosulfan with adverse health impacts in Kerala, and ensure its continued use. They have suppressed facts by sponsoring fraudulent reports and have manipulated government committees. They have vilified and threatened organisations and people who have researched, written about or spoken against endosulfan. They have filed cases against scientists who have conducted research on endosulfan.

In 2006, after the Union ministry of agriculture banned the use of endosulfan in Kerala, pesticide manufacturers set up a not-for-profit organisation called Centre for Environment and Agrochemicals. This body claimed to be working for the welfare of farmers and promoted what it called a judicious use of pesticides. One of its objectives, it claimed, was to expose scientifically fraudulent reports about pesticides.

This industry-supported organisation began harassing insititutions and individuals who had supported the ban. It filed defamation cases against

CSE and anyone who spoke at CSE's conferences or in anti-endosulfan meetings. It distributed booklets with obscene cartoons near the CSE office. In 2007, its hired hands demonstrated in front of the CSE office and the residence of CSE's director general, Sunita Narain.

Another similar body, the Crop Care Federation of India (CCFI), filed defamation cases against CSE and lost twice. The first case was filed in 2006 and lost in 2008. The CCFI challenged this order by filing a criminal revision application, which was rejected as the sessions judge ruled that the CSE report was not defamatory in nature.

9. *Has the ban on endosulfan affected Indian agriculture?*

CSE analysed the situation nine months after the ban and found that there were no reports of crops being devastated by pests or massive reductions in food production due to the lack of endosulfan – a scenario which was being painted by endosulfan manufacturers. Farmers have replaced endosulfan with other pesticides. For instance, in Vidarbha, Maharashtra, it was reported that farmers were getting along very well without endosulfan. Most farmers had simply shifted to the slightly more expensive quinalphos, while a small number had started looking for organic alternatives.

GLOBAL STATUS OF ENDOSULFAN BAN

"The nationwide benefits to society as a whole are low for all uses of endosulfan and do not exceed the risks."
"EPA has concluded that endosulfan poses unacceptable risks to agricultural workers and wildlife, and can persist in the environment."
US Environmental Protection Agency

"Persistence of 'endosulfan' coupled to ongoing use of endosulfan pesticides, will ensure continued long-range transport and contamination of remote environments."
"Endosulfan fulfils all of the hazard criteria for classification as a POP under the UN-ECE Protocol on POPs and the UNEP Stockholm Convention."
'Endosulfan, a global pesticide: A review of its fate in the environment and occurrence in the Arctic', Science of the Total Environment 2010; 408:2966-2984

"A primary risk assessment concern for endosulfan is that effects resulting from exposure during critical development stages (*in utero*, or to infants and children) will result in endocrine disruption and subsequent neurotoxicity, developmental or reproductive adverse effects that are irreversible."
'Human health risk assessment of endosulfan. I: Toxicology and hazard identification', Regulatory Toxicology and Pharmacology 2010; 56:4-17

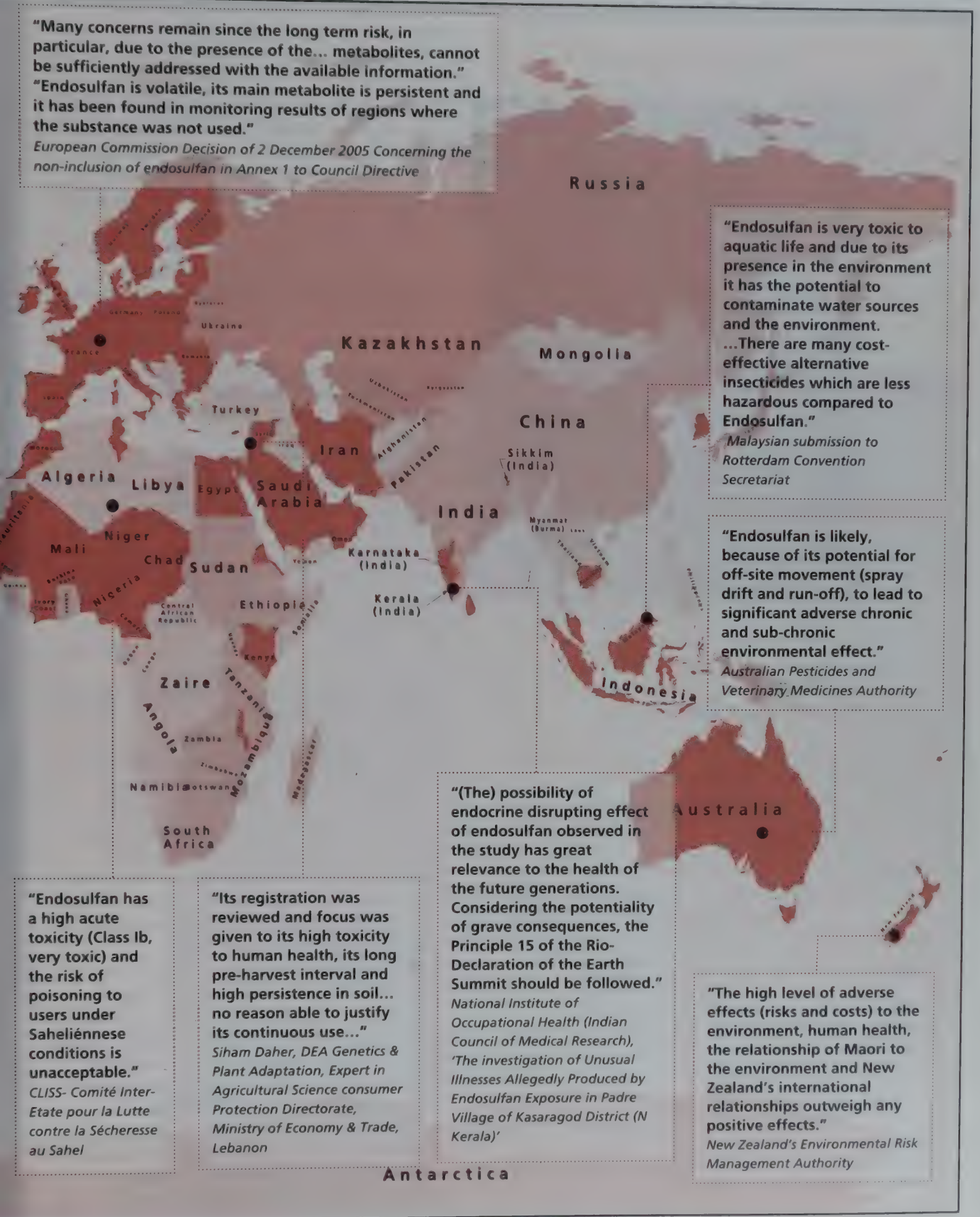
"According to Anvisa, the measure is based on toxicological studies linking pesticide application to endocrine and reproductive problems in farm workers and the population in general."
Estado. Anvisa: Agência Nacional de Vigilância Sanitária, Brazil

"The resolution's main objective is to protect human health, animal and environmental conservation, taking into account that (endosulfan) is a highly toxic insecticide."
El Servicio Nacional de Calidad y Sanidad Vegetal y de Semillas (SENAVE), Paraguay

Status of endosulfan in countries

- Banned or phasing out
- Not permitted, but not banned
- Legally using with no phase-out
- Not using, or unknown status

Source: PEN (International POPs Elimination Network) and ACAT (Alaska Community Action on Toxics)


"Many concerns remain since the long term risk, in particular, due to the presence of the... metabolites, cannot be sufficiently addressed with the available information." "Endosulfan is volatile, its main metabolite is persistent and it has been found in monitoring results of regions where the substance was not used."
European Commission Decision of 2 December 2005 Concerning the non-inclusion of endosulfan in Annex 1 to Council Directive
Russia
"Endosulfan is very toxic to aquatic life and due to its presence in the environment it has the potential to contaminate water sources and the environment. ...There are many cost-effective alternative insecticides which are less hazardous compared to Endosulfan."
Malaysian submission to Rotterdam Convention Secretariat
Kazakhstan
Mongolia
China
Turkey
Iran
Afghanistan
Pakistan
Sikkim (India)
Algeria
Libya
Egypt
Saudi Arabia
India
Myanmar (Burma)
Niger
Mali
Chad
Sudan
Karnataka (India)
Kerala (India)
Nigeria
Ethiopia
Central African Republic
"Endosulfan is likely, because of its potential for off-site movement (spray drift and run-off), to lead to significant adverse chronic and sub-chronic environmental effect."
Australian Pesticides and Veterinary Medicines Authority
Indonesia
Zaire
Tanzania
Angola
Zambia
Mozambique
Namibia
Botswana
Madagascar
South Africa
"(The) possibility of endocrine disrupting effect of endosulfan observed in the study has great relevance to the health of the future generations. Considering the potentiality of grave consequences, the Principle 15 of the Rio-Declaration of the Earth Summit should be followed."
National Institute of Occupational Health (Indian Council of Medical Research), 'The investigation of Unusual Illnesses Allegedly Produced by Endosulfan Exposure in Padre Village of Kasaragod District (N Kerala)'
Australia
"Endosulfan has a high acute toxicity (Class Ib, very toxic) and the risk of poisoning to users under Saheliénnese conditions is unacceptable."
CLISS- Comité Inter-Etate pour la Lutte contre la Sécheresse au Sahel
"Its registration was reviewed and focus was given to its high toxicity to human health, its long pre-harvest interval and high persistence in soil... no reason able to justify its continuous use..."
Siham Daher, DEA Genetics & Plant Adaptation, Expert in Agricultural Science consumer Protection Directorate, Ministry of Economy & Trade, Lebanon
"The high level of adverse effects (risks and costs) to the environment, human health, the relationship of Maori to the environment and New Zealand's international relationships outweigh any positive effects."
New Zealand's Environmental Risk Management Authority
Antarctica

1.3 PESTICIDES IN BLOOD

PHOTOGRAPHS SURYA SEN / CSE

Train No 339 leaves Abohar in Punjab every night to reach Bikaner in Rajasthan the next morning. Local people refer to it as the 'Cancer Train' – nearly 100 cancer patients travel by it every day for diagnosis and treatment at the Acharya Tulsi Regional Cancer Treatment and Research Institute in Bikaner.

Years of pesticide use and misuse are taking their toll on India's green belts, and Punjab occupies a 'place of pride' amongst them. Studies indicate that pesticide use in the cotton-growing state might be linked to the rising cases of various kinds of cancer there. Ferozepur, Faridkot, Moga, Mansa, Barnala, Bathinda... the list of districts from where the dead and the dying are coming is growing.

In 2005, CSE decided to investigate: it found a cocktail of 6-13 different pesticides in each of the 20 blood samples tested from four villages in Punjab. And came up against innumerable questions – how much of pesticides in our blood can be considered 'safe'? Does a safety threshold level exist? If yes, how do scientists and the industry compute it? As we dug deeper, it became clear that science claims much, but understands little.

1. What prompted CSE to test the blood of Punjab farmers for pesticides?

Punjab is synonymous with the Green Revolution. But this revolution also ushered in a boom time in pesticide use: consumption of pesticides in the country increased from 154 metric tonne (MT) in 1954 to a peak of 88,000 MT in 2000-2001. Punjab was the second highest user in terms of total pesticide consumption and the highest user in terms of per capita or per hectare use.[1] In 2009-10, of the 41,644 MT of pesticides consumed in the country, Punjab alone accounted for 5,810 MT.

A large proportion of pesticides in India is used for cotton and rice cultivation. Excessive use of pesticides in these two crops in Punjab is something that has been well documented. The Ludhiana-based Punjab Agricultural University recommends only seven sprays on cotton in six months, but farmers in Bathinda spray as many as 32 times within the same period![2]

There is a lack of awareness about the use and disposal of pesticides; people often use empty pesticide containers for storing food and water. This rampant and careless use has had deadly results.[3]

Studies indicate the presence of pesticide residues in breast milk, bovine milk, fruits and vegetables from Punjab; reports of high incidence of cancer have been emerging from certain areas as well.

In 2004, the Punjab Pollution Control Board (PPCB) commissioned the School of Public Health (SPH) at the Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh to do a year-long study to explore high cancer rates in certain areas of Bathinda district. Though it did not directly link cancer cases with pesticides, the study did find high cancer rates in Bathinda, where pesticide usage is very high (see Box: *An epidemiological study*).

In 2005, after the PGIMER report was published, NGOs from Punjab approached CSE to conduct further investigations on the linkages between pesticide usage and cancer in the cotton belt of Punjab.

2. *What was the CSE study?*

In 2005, CSE's Pollution Monitoring Lab randomly collected and analysed 20 blood samples from four different villages of Punjab: Mahi Nangal, Jajjal and Balloh in Bathinda district and Dher in Ropar district. The villages were selected based on the feedback from the NGOs, which said that these villages had higher pesticide usage and high cancer incidence. The blood samples were analysed for 14 organochlorines and 14 organophosphorus pesticides following methodology based on USEPA protocols.

AN EPIDEMIOLOGICAL STUDY

The 2005 report of the School of Public Health (SPH) at the Post Graduate Institute of Medical Education and Research (PGIMER) in Chandigarh, titled 'An epidemiological study of cancer cases reported from villages of Talwandi Sabo block, district Bathinda, Punjab', compares cancer incidence in Talwandi Sabo (referred to in the report as the study area) with that in Chamkaur Sahib block (described by the report as the control area) of Roop Nagar district, Punjab.

Farmers in Talwandi Sabo use canal water and cultivate cotton, while their counterparts in Chamkaur Sahib use groundwater and cultivate rice or wheat. The rationale for selecting areas with different irrigation patterns was to ascertain links between canal irrigation and cancer. The study also tried to find if cotton cultivation – much more pesticide-dependant compared to rice and wheat – could be linked to cancer.

A total population of 1,83,243 consisting of 39,732 families in 129 villages was surveyed: it included a population of 85,315 in 36 villages of Talwandi Sabo and a reference population of 97,928 in 93 villages of Chamkaur Sahib. A total of 7,441 deaths were recorded which occurred in 10 years (1993-2003).

Age-adjusted cancer death rate per 1,00,000 population per year at Talwandi Sabo was 51.2, while in Chamkaur Sahib, it was 30.3. Age-adjusted prevalence of confirmed cancer cases was 103 per lakh at Talwandi Sabo and 71 per lakh in Chamkaur Sahib. Cancers of the female reproductive system – breast, uterus/cervix and ovary – were more common in Talwandi Sabo, whereas cancers of blood and lymphatic system, esophagus and bones were more common in Chamkaur Sahib block.

Significantly, 63.8 per cent of the cropped area in Talwandi Sabo was under cotton (which was not cultivated at all in Chamkaur Sahib). More than half of all the pesticides manufactured in the country are used in cotton. The SPH study shows that many more people sprayed pesticides at Talwandi Sabo as compared to Chamkaur Sahib.

The SPH tested water (tap and ground) and vegetables from the study and control areas. For the month of March 2004, heptachlor levels in tap water of Talwandi Sabo were 0.00004 parts per million (ppm) – higher then the permissible limit (0.00003 ppm); this pesticide was not found in Chamkaur Sahib tap water. The April samples for groundwater showed similar results: hepatchlor levels of Talwandi Sabo were in the range of 0.006 ppm, while the pesticide was not present in Chamkaur Sahib. Malathion in Talwandi Sabo's tap water samples of April 2004 was 0.21 ppm: 420 times higher than the permissible limit (0.0005 ppm). The pesticide was within the permissible limit in Chamkaur Sahib (0.00009 ppm). "The levels of heptachlor, a known carcinogen that is banned in India, were higher in samples of tap water in Talwandi Sabo compared to Chamkaur Sahib," concludes the report. "The same holds for groundwater too," it states.

The report, however, observes: "It's difficult to pinpoint a single cause for cancer... A multi-pronged strategy to provide safe water supply, discouraging indiscriminate pesticide use... is recommended."[1]

STUDY HIGHLIGHTS

CSE found 15 different pesticides – a cocktail of 6-13 pesticides, all different, in the 20 blood samples tested from four villages in Punjab.

Organochlorine pesticides

- Samples tested for 14 organochlorine pesticides.
- 11 of the 14 pesticides were detected.
- α, β, γ isomers of hexachlorocyclohexane (HCH) were detected in 95 per cent, 35 per cent and 100 per cent of the samples, respectively.
- Aldrin was detected in 80 per cent of the samples; other pesticides found were heptachlor (5 per cent), chlordane (70 per cent), DDD (55 per cent), DDE (95 per cent), DDT (50 per cent), a-endosulfan (24 per cent) and b-endosulfan (5 per cent).
- Mean total DDT in blood samples of Punjab was 0.065
- Mean levels of aldrin were 0.0062 mg/l and ranged fr detectable (ND) to 0.0159 mg/l.
- Mean levels of alpha and beta endosulfan were 0.00 0.0002 mg/l, respectively. Mean total endosulfan samples was 0.0046 mg/l.
- HCH, detected in all the samples, had a mean level of mg/l.

Organophosphate pesticides

- Samples tested for 14 organophosphate pesticides.
- 4 of 14 pesticides were detected.
- Monocrotophos was found in 75 per cent of the sar phosphamidon in 70 per cent, chlorpyrifos in 85 per ce

TEST RESULTS

RESIDUES (mg/l in whole blood)	ORGANOCHLORINES												
	α-HCH	β-HCH	t-HCH	δ-HCH	t-HCH	Heptachlor	Aldrin	Chlordane	DDE	DDD	DDT	t-DDT	α-Endosulfan
Total no of individuals	20	20	20	20	20	20	20	20	20	20	20	20	20
No of samples in which pesticide detected	19	7	20	0	20	1	16	14	19	11	10	19	5
Percentage tested positive	95	35	100	0	100	5	80	70	95	55	50	95	25
Mean (mg/l)	0.0281	0.0063	0.0227	0.0000	0.0570	0.0006	0.0062	0.0090	0.0450	0.0102	0.0100	0.0652	0.0044
Median (mg/l)	0.0136	0.0000	0.0190	0.0000	0.0542	0.0000	0.0059	0.0028	0.0365	0.0065	0.0027	0.0526	0.0000
Minimum (mg/l)	0.0000	0.0000	0.0136	0.0000	0.0241	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
Maximum (mg/l)	0.1054	0.0382	0.0569	0.0000	0.1279	0.0110	0.0159	0.0539	0.2554	0.0473	0.0485	0.2693	0.0539

Notes:
1. t–HCH = α + β + δ HCH, t-DDT-DDD+DDE+DDT, t- endosulfan- α + β + endosulfan sulfate
2. Dieldrin, Dichlorvos, acephate, phorate, diazinon, dimethoate, fenthion, quinalfos, phenthoate, profenofos and ethion were not detected in any of the samples.

3. What were the findings of the CSE study?

The study found residues of 6-13 pesticides in each of the blood samples. The pesticide cocktail included old and persistent pesticides like DDT and lindane, as well as highly toxic ones like monocrotophos and chlorpyrifos (see Box: *Study highlights*). Studies done across the world indicate a strong linkage between these pesticides and chronic diseases (see Box: *Health impacts of pesticides found in blood of Punjab farmers*).

The levels of some of the persistent organochlorine pesticides (OCs) found in the samples were astounding: 15-605 times higher than those found in the blood samples of people in the US, tested by the US Centre for Disease Control and Prevention (see Box: *Comparing contamination*).

The CSE study detected hexachlorocyclohexane (HCH) in all the blood samples, and DDT in 95 per cent of the samples. The study was one of the first in India to test for organophosphate pesticides (OPs) in human blood – these were found in equally high levels. Industry claims that these pesticides are not persistent and will degrade quickly. But monocrotophos was detected in 75 per cent of the blood samples, while chlorpyrifos was present in 85 per cent. Seventy per cent of the samples also contained two more OPs: phosphamidon and malathion.

malathion in 70 per cent, indicating regular and widespread exposure to these pesticides.

Mean level of monocrotophos in blood samples was 0.0948 mg/l, in the range of ND to 0.4915 mg/l.

Mean level of chlorpyrifos was 0.0662 mg/l and ranged from ND to 0.4965 mg/l.

Mean level of phosphamidon was 0.0366 mg/l and ranged from ND to 0.1282 mg/l.

Malathion was detected in whole blood samples from Punjab at a mean level of 0.0301 mg/l, and ranged from ND to 0.0753 mg/l.

The collected data indicated that each person CSE tested was exposed and carried a body burden of multiple pesticides. This presence of esticide residues might be due to direct exposure during application of esticides, and/or due to exposure through air, water, and food.

	ORGAN OPHOSPHORUS							
	t-endosulfan	Total organo chlorines	Monocrotofos	Phosphamidon	Chlorpyrifos	Malathion	Total organo phosphorus	Total pesticides
0	20	20	20	20	20	20	20	20
	6	20	15	14	17	14	17	20
	30	100	75	70	85	70	85	100
000	0.0046	0.1424	0.0948	0.0366	0.0662	0.0301	0.2278	0.3701
000	0.0000	0.1185	0.0517	0.0321	0.0358	0.0273	0.1924	0.3290
000	0.0000	0.0497	0.0000	0.0000	0.0000	0.0000	0.0000	0.1224
000	0.0539	0.3252	0.4915	0.1282	0.4965	0.0753	0.6409	0.7627

Shockingly, the average levels of monocrotophos in the Punjab blood samples (0.095 ppm) were found to be four times higher than the short-term exposure limit for humans set by the World Health Organization/Food and Agricultural Organization. If we consider the long-term exposure limits, the results are even more unnerving: the average amount of monocrotophos in the blood of the population CSE tested was 158 times higher than the long-term exposure limit for humans!

4. *What is the 'safe level' of pesticides in our bodies?*

There are no safety norms for pesticides in our bodies – in body fluids like blood or in tissues. Science is not able to establish direct linkages between the levels of pesticides and toxins in our body and the resultant health impacts. What science does establish is toxicity of individual pesticides – a dose of pesticide necessary to produce a measurable harmful effect. This is usually established through tests on mice, rats, rabbits and dogs. Results are then extrapolated on humans, and safe exposure levels predicted.

Two types of toxicity are established: acute toxicity and chronic toxicity.

Acute toxicity of a pesticide refers to the ability of the chemical to cause injury to a person or animal from a single exposure, generally of a short duration. The value commonly used to measure acute toxicity is LD 50 (a lethal dose in the short term; the subscript 50 indicates the dose is toxic enough to kill 50 per cent of lab animals exposed to the chemical). LD 50 values of pesticides are recorded in milligrams of pesticide per kilogram of body weight and are measured zero onwards; the lower the LD 50, the more acutely toxic is the pesticide. Parathion, for example, is considered to be highly toxic because the oral lethal dose is less than 4 milligram per kilogram (mg/kg) of body weight, compared with 1,200 mg/kg for malathion, or 5,000 mg/kg for methoxychlor.

Chronic toxicity is the harmful effects that occur from small doses repeated over a period of time, usually years. To establish chronic toxicity, animals receive a pesticide-laced diet from a very young age. This continues till they show chronic adverse affects, whether carcinogenic or mutagenic in nature, or with definite impairment in their reproductive systems. Such experiments have a purpose: determine a 'No Observable Effect Level' (NOAEL) of pesticide exposure – a level in the total diet that causes no effect on test animals as compared to others maintained under unexposed conditions. Sometimes, it is not possible to deduce this number. Then, the safety mark is established at the point where the first, minutest, adverse effect appears. This is called LOAEL, or the 'Lowest Observable Adverse Effect Level'.

Some of the chronic effects found in test animals exposed to certain pesticides include birth defects (teratogenesis); toxicity to a fetus (fetotoxic effects); production of tumors (oncogenesis) either benign (noncancerous) or malignant (cancerous/carcinogenesis); genetic changes (mutagenesis); blood disorders (hemotoxic effects); nerve disorders (neurotoxic effects); endocrine disruption; and reproductive effects. The NOAEL/LOAEL is expressed as pesticide exposure in

COMPARING CONTAMINATION: CSE AND CDC

The US-based Centre for Disease Control and Prevention (CDC) regularly conducts one of the most comprehensive bio-monitoring programmes in the world: the National Report on Human Exposure to Environmental Chemicals. This programme aims to determine the chemical body burden of US residents and also investigate potential links to diseases.

The CDC's second report of January 2003 contained an analysis of blood and urine levels of 116 environmental chemicals. The sample population was studied from 1999 through 2000.

Five of the pesticides CSE tested for were those studied by the CDC – and the Punjab samples had far higher pesticide residues. For example, lindane residues in CSE's samples were 600 times higher than in the CDC study; levels of DDE and DDT in the Punjab samples were 35 times and 188 times higher respectively than in the US samples.

	b-HCH	Lindane	Heptachlor	DDE	DDT
PUNJAB BLOOD SAMPLES					
Percentage of sample with pesticides (%)	35	100	5	95	50
Mean concentration of pesticide (ng/g of lipids)	1,254	4537	110	8,996	1,990
Pesticides found in blood by CDC study on US population	100	0.0005			
Percentage of sample with pesticides (%)	62	1.7	27	99	26
Mean concentration of pesticide (ng/g of lipids)	9.68	<7.5	<7.5	260	<10.6
Number of times the Punjab samples exceed the pesticides concentration when compared to US samples	129	605	15	35	188

Note: Organochlorine pesticides are lipophilic and concentrate in the body's lipid stores including the lipid in the blood serum. For comparison, levels for these compounds are expressed as per gram of total lipid in the blood serum.

milligram per kilogram of body weight per day (mg/kg bw/day).

LD 50 and NOAEL values are then extrapolated to determine safety values for humans, known as acute reference dose (aRfD) for acute toxicity and acceptable daily intake (ADI) for chronic toxicity. aRfD and ADI are arrived at by adjusting LD 50 and NOAEL values downwards, usually by a factor of 100, respectively. A division factor of 10 is used to allow for the possibility that humans are more sensitive than animals; another division factor of 10 is used to allow for differences amongst individual humans. There is a growing demand to include another division factor of 10 to account for cumulative impacts of multiple pesticides now being found in human beings.

aRfD is used to set occupational exposure standards for pesticides. ADI is used to set pesticide standards on food and water. The scientific assumption is that if the daily consumption of pesticides is below the ADI value, then it is safe. However, this assumption discounts the accumulation of pesticides in our bodies and the resultant health impacts.

5. What do we know about pesticides in our bodies and their health impacts?

Very little is known about the link between pesticide body burden and health impacts. Biomonitoring studies – measuring chemicals in blood, urine, breast milk, fat, hair or other tissues – do reveal that large amounts of synthetic chemical residues have infiltrated our bodies. Yet, industry argues there is no evidence these chemicals cause any harm. Having chemicals in our bodies is unavoidable in modern times, it contends. It takes refuge behind what it calls a lack of 'authentic' epidemiological studies correlating disease to the invasion by a pesticide. Industry also finds it useful that there are multiple pesticides and chemicals in our bodies. This makes it difficult to pinpoint blame. But toxicological impacts of individual pesticides is stark (see Box: *Health impacts of pesticides found in blood of Punjab farmers*). Also, there is no evidence that proves the safety of carrying a chemical body burden.

But there is a growing scientific opinion that finding the exact effects of chemical body burden is not the issue at all: identifying the cocktail of chemicals in human bodies and setting up regulatory systems to reduce the chemical body burden is more critical. The general premise is that, irrespective of their impacts, synthetic chemicals should not be allowed to trespass human bodies.

6. What is the world doing to reduce chemical body burden?

Many researchers, scientists and NGOs now believe there should be a paradigm shift in the way

HEALTH IMPACTS OF PESTICIDES FOUND IN BLOOD OF PUNJAB FARMERS

Cancer: Evidence of the links between pesticide exposure and breast cancer is mixed, with many studies showing no co-relation and others showing strong linkages. In a recent study it was shown that a mixture of four organochlorines (pp' DDT, pp' DDE, b-BHC and pp' DDT) acted together to trigger growth of breast cancer cells.

Malathion has been shown to induce changes in the epithelium of rat mammary glands, influencing the process of carcinogenesis. Multiple studies have shown that farmers are more likely to develop leukemia, brain, prostrate and skin cancer and non-Hodgkin's lymphoma than the general population.

Lymphoma: In-utero and early childhood exposure to pesticides is associated with a significantly increased risk of developing non-Hodgkin's lymphoma (NHL), a cancer that begins in the cells of the lymph system (which includes the spleen, thymus, tonsils, bone marrow, lymph nodes and circulating white blood cells, called lymphocytes). The lymph system is part of the immune system, which protects the body from disease and infection. NHL is characterised by the excessive accumulation of atypical lymphocytes, which crowd the lymph system and suppress the formation and function of blood and immune cells. This leads to a diminished ability of the body to fight infection. Studies indicate an association between pesticide exposure and NHL in children exposed to lindane, DDT, and organophosphorus pesticides.

Mutagenesis (genetic changes): Pesticides have been shown to cause genetic changes – DDT and its metabolites can induce DNA damage in peripheral blood mononuclear cells. A significant correlation between levels of DDT, DDD, DDE in the blood and DNA damage was found in women with different amount of environment exposure to DDT and its metabolites.

Monocrotophos is known to induce single/double strand DNA breaks in mice. Malathion can cause DNA abnormalities at all doses (0.02, 0.2, 2 and 20 g/l) when added to human blood cells. Studies suggest that malathion is a mild mutagen and at higher concentrations, might cause genotoxicity in humans.

Neurotoxicity (toxicity of the brain or nervous system): Chlorpyrifos, one of the most widely used organophosphorus pesticides, has been reported as a developmental neurotoxin specifically targeting the immature brain and increasing its vulnerability.

Exposure can result in adverse effects on brain cell development. Tests conducted on neonatal rats indicated development of behavioral deficits in adolescence and adulthood.

Parkinson's disease: Epidemiological studies have suggested an etiologic relationship between pesticide exposure (of dieldrin, for instance) and Parkinson's disease.

Fetotoxicity (toxicity to fetus): A strong relationship has been found between prematurely delivered and low birth weight babies and the level of DDE, a metabolic breakdown product of DDT, in their mothers. Studies carried out on rats showed that repeated exposure to sub-threshold doses may lead to growth retardation, behavioral abnormalities and muscle weakness. Studies also indicate fetotoxic and teratogenic effects at a maternal dose of 25 mg/kg per day, a dose that also produced maternal toxicity.

Teratogenic (birth defects): Children born to women who live in a high pesticide-use area while pregnant face an increased risk of birth defects – cleft lip, limb reduction defects and neural tube defects. Recent findings suggest that chlorpyrifos, a suspected neuroteratogen, impacts the development of neurons.

Malathion has been shown to cause birth defects in a variety of wildlife, at levels lower than other pesticides. When administered to adult animals, malathion leads to muscle weakness and paralysis. Neonates (newborn babies) are far more sensitive to these agents than adults, mainly because of a slower rate of detoxification of the metabolite (the metabolite in this case would be malaoxon, which has been shown to be far more toxic than malathion itself).

Immunological changes: Studies have found that farmers or farm hands in the former German Democratic Republic carried traces of more than one chemical – most commonly DDE, PCBs, and HCB; 80 per cent of them had been exposed for more than 20 years which had resulted in frequent infections and immunological abnormalities. Chronic exposure to chlorpyrifos has been shown to cause immunological change.

Decreased sperm quality and quantity: Some studies link pesticide exposure with decreased sperm quality, and vice versa (a higher sperm density with lower pesticide exposure). Hormone disruption is considered a possible contributor to low sperm count and dozens of pesticides are known or suspected hormone disrupters. Lindane was found to be estrogenic to female rats and mice, and also caused the testes of male rats to become atrophied. Seminiferous tubules and Leydig cells (important for production of sperms) were completely degenerated at doses of 8 mg/kg/day over a 10-day period.

Treatment with 1-40 mg of lindane/kg of body weight disrupts testicular morphology and leads to other disorders.

BHOLA SINGH IS DEAD

Bhola Singh was a farmer from village Balloh, district Bhatinda, Punjab. He is part of a story with a beginning and an end. But it has no middle. Thus what follows is a fragment. But it is not fiction.

Punjab practices intensive agriculture that needs pesticides. Industry says that, compared to the developed world, we use little pesticide in India. That is true, perhaps even in Punjab. But what industry does not tell you is that we find much more pesticide, compared to the developed world, in our food, our water, our soils. And now, our blood. As you will find, in this story that has no middle.

We know pesticide use in Punjab is one of the highest in the country. We also know there are residues in the food it produces. But what else? Do we know what pesticides are doing to people there? Whether cancer rates there are higher, or of a correlation between growing disease burden and the use of toxins? No. We don't. Why? Because there is no definite evidence. So, industry says, don't ask hypothetical questions. So, this story has no middle.

But our colleagues have tried to understand the science and politics of pesticide regulation: a piece of the missing middle. Another piece: they travelled to two districts – among the highest users of pesticides in Punjab – and collected samples of human blood. This was in October, 2004: spraying season. Pesticides everywhere: in fields and storerooms of kitchens. Shops stocked to the rafters. Pesticides sold over the counter, like aspirin. You can mix, you can match, use as much as you can afford. There are no rules here. No corporate responsibility on educating farmers on right use. But the market works on marketing, not on telling the truth, right? Nobody is supposed to ask what is right. Because there is nobody to tell you what is wrong.

This story tries that. We found pesticides in the blood samples. All samples. A mixture of pesticides. At high levels. And at this point, the story falls apart again. For, what is a 'high level' in human blood? How much is safe in human blood? How much unsafe? We do not know. There exist studies in India, of human blood, for a range of what are called 'persistent' pesticides; they bioaccumulate. But such pesticides have been banned, at least on record. So are the residues we found a 'historical burden'?

No. Moreover today, says industry, they peddle Gen-next pesticides: these do not persist. They degrade. We beg to differ, strongly. Invented for chemical warfare during World War II, these Gen-next concoctions are less persistent but more toxic. We found the blood of people in Punjab contain these pesticides, which should have disintegrated into metobolites and excreted, as industry would have it. But they are there. We found them.

Industry is completely mum on what Gen-next pesticides do, in the human body, in the time they circulate there. And what about metabolites? What do we know about their impact on our bodies?

We know chemicals can suppress the immune system, in turn triggering disease. But since no doctor will give you a death certificate which says the cause is an unknown trigger, industry is safe. We cannot pinpoint blame. All stories of cancer will be dismissed as fiction. No truth. No connection. Certainly no liability.

But this is chemical rape. What we found in Punjab has no comparison. Still, we are not saying cancer in Punjab is because of the pesticide it uses. There is no proof. In this world of industry, the onus is on us to prove, our death, in the face of an evident murderer.

So, we ask you to read. Do read between the lines, the horrendous pain of cancer and no cause. We ask you to read so that we can jointly ask for change. This, we repeat, is not fiction. It is real life. About all of us. About our bodies. It cannot go unsaid. It will not remain denied.

Sunita Narain and Chandra Bhushan, *Down To Earth*, June 15, 2005

pesticides are regulated. It is no more about monitoring pesticides in food commodities only. It is about checking the body burden and then regulating these toxins.

Body burden studies hold the key to a fool-proof system to regulate the use of pesticides and other chemicals. This view is also gaining ground among policymakers in the West – albeit slowly. For example, in 2003, the UK Royal Commission on Environmental Pollution stated, "where chemicals are found in elevated concentrations in biological fluids such as breast milk, they should be removed from the market immediately". In fact, policymakers in New Zealand and the state of Tasmania in Australia are now debating chemical trespass bills which will render illegal the ingress of agricultural chemicals into the human body and also facilitate the recovery of damages.

Similar concerns are also driving action elsewhere. On April 27, 2003 the US Supreme Court affirmed the rights of consumers, workers and farmers to sue pesticide manufacturers when their product causes harm. The ruling strikes down the pesticide industry's persistent claims that registration of a pesticide under the US federal pesticide law automatically shields manufacturers from damage claims. The judgement also overrules the pesticide industry's claims that the Federal Fungicide Insecticide and Rodenticide Act, 1948 (thereafter amended many times) pre-empts local and state liability statutes.

Assigning primary responsibility to pesticide manufacturers is an efficient way to address the problem of chemical trespass. So far, little penal action has been possible against these manufacturers, even when a clear link has been established between their product and its adverse health implications. To be effective, the modern regulatory system has to consider the chemical body burden and hold manufacturers of these toxins accountable.

7. *What is India doing about chemical body burden?*

Biomonitoring has not yet been institutionalised in India; the government has no specific programme for it. However, a few public as well as private institutions have conducted tests to find pesticide residues in human blood, fat and milk. In the past, Indian researchers have found DDT and BHC levels in the blood of Indians to be among the highest in the world. For example, a 1992 study published in the

journal, *Science of Total Environment* found DDT levels to be as high as 7.17 parts per million in blood samples. However, most Indian studies on pesticide residues in humans are restricted to older organochlorine pesticides such as DDT and BHC. We have not come across any research on the presence of OPs in the blood of Indians.

Clearly, the new burden that modern India is carrying – as Punjab clearly exemplifies – needs to be examined by its scientists. Doing anything less would be negligent.

8. What steps has the Punjab government taken to reduce its body burden of pesticides?

After CSE raised the issue in 2005, the Punjab government constituted two committees – a high-powered committee and an expert group. While the expert group held one meeting, the high-powered committee had none. The state government also ordered a study and immediate health remediation measures; the government decided to formulate an organic farming policy for the state as well, but not much has happened in that quarter either.

In 2009, a house-to-house survey was conducted by the state health department. The prevalence of cancer in Punjab as per the survey was 30.54 per lakh population, whereas the prevalence in India is 125 per lakh, according to the state data. The worst hit is the Malwa belt, with Muktsar and Bathinda reporting the maximum incidence of cancer at 75.1 and 75 patients per one lakh population, respectively: more than double the state average. The lowest incidence of 10.8 is in Amritsar.

The state has begun a Cancer Registry Program; this followed the approvals given by the Indian Council of Medical Research (ICMR) to the Government Medical College, Patiala for Population-based Cancer Registry (PBCR) and to PGI, Chandigarh for Hospital-based Cancer Registry (HBCR). The PBCR, set up in Bathinda, is supposed to design suitable treatment facilities.

What is quite clear is that after 2005, awareness about pesticide misuse and its possible linkages with rising cancer cases has increased significantly in Punjab. Civil society groups are actively promoting non-pesticidal agriculture and political parties have started talking about this problem in their election manifestoes. The 2012 election manifesto of the Congress party states: "Punjab is suffering from ecological mess due to excessive use of pesticides and fertilisers on its farms." However, the manifesto does not promise any solution for this 'mess'.

What Punjab needs is new paradigm – an ecological and health-based agriculture paradigm.

1.4 PESTICIDES IN CARBONATED BEVERAGES

The Indian parliament constitutes Joint Parliamentary Committees (JPC) only on critical issues of public interest. Of the five JPCs constituted in the history of independent India, one was set up in August 2003 to investigate the issues arising out of CSE's study on pesticide residues in soft drinks.

In 2003, CSE tested various soft drink brands and found them to be contaminated with high levels of pesticides. This led to an uproar in the country; many states banned these sugary drinks and the Indian parliament stopped their sales in its canteen. CSE demanded a strict mandatory standard to check pesticide residues in these drinks. The JPC was set up.

Interestingly, the terms of reference (ToR) of the JPC were not to investigate the companies but to check "whether the recent findings of CSE regarding pesticide residues in soft drinks are correct or not". Another ToR was to suggest criteria for evolving suitable safety standards for soft drinks, fruit juices and other beverages where water is the main constituent.

In February 2004, the JPC recommended setting strict pesticide standards for soft drinks. For the next two years, the government went around setting up one committee after another, but did not notify any standards. Keeping this inaction in mind, CSE released another study on pesticides in soft drinks in August 2006. This time, the samples were collected from across the country. This study too found very high levels of pesticides. Another round of confrontation ensued between the government, CSE and industry.

Finally, in August 2008, the Union health ministry notified the rules for maximum levels of pesticides in carbonated water. It specified 1 ppb (parts per billion) as the maximum permissible limit for lindane, DDT, malathion and five other pesticides. This is the world's first standard on pesticides in soft drinks.

CSE accepted this standard as an interim standard, as it believed that the draft standards set by the Bureau of Indian Standards (BIS) (which is 10 times stricter than the mandatory health ministry standards) were more appropriate to control pesticides in these sugary drinks. The fight to make the BIS standard mandatory continues.

1. *What prompted CSE's first study on pesticide residues in soft drinks?*

Following our bottled water study in 2003 (see Box: *Bottled water came first*), people wrote to us from across the country, to know whether other products that used water as a raw material – such as soft drinks – were also contaminated.

We were intrigued by the government's response to the bottled water study: it was prevaricating on legislating the new stringent norms for bottled water. Who was the government trying to protect? What was at stake – the Rs 1,000-crore bottled water business, or the much larger Rs 7,000-crore soft drink industry? This made us check soft drinks for pesticide residues.

2. *What did the study find?*

In 2003, CSE's Pollution Monitoring Lab tested 12 brands of soft drinks sold in Delhi. These were tested for 16 organochlorine pesticides, 12 organophosphorus pesticides and four synthetic pyrethroids – all commonly used in India in agricultural fields as well as in homes. As Coca-Cola and PepsiCo controlled 90 per cent of the soft drinks market in India, most of the brands that were tested were theirs. These brands included Pepsi Cola, Mountain Dew, Diet and Blue Pepsi, Mirinda, 7-Up, Coca Cola, Fanta, Limca, Sprite and Thums-Up.

All samples of the 12 brands contained poisonous pesticide residues (lindane, DDT, chlorpyrifos and malathion). The average residues in the samples were found to be 11-70 times the European Union (EU) norm for pesticide residues in drinking water (0.0001 mg/l or 0.1 ppb). Mirinda Lemon, Coca Cola and Pepsi Cola carried the greatest amounts of residues. The residues were measured against the EU norm because (a) India did not have pesticide residue standards and (b) EU norms covered all pesticides. No other country in the world had standards for all the pesticides being used in India.

3. *What was the immediate fallout of the study? How did the government and industry respond?*

The first response to CSE's study findings came from the two soft drink giants, which did an unprecedented joint press conference junking the CSE study. They questioned the study methodology, the capacity and credentials of the CSE lab to do such a study, and even CSE's motives. The issue came up in the Lok Sabha, which banned soft drinks from the Parliament premises, while the then Union health minister ordered testing of soft drinks in two government labs. These labs found pesticide residues, but in lower quantities than those detected by CSE. In August 2003, a Joint Parliamentary Committee (JPC), under the chairmanship of Sharad Pawar, was set up to look into the matter. It upheld CSE's findings and made a wide range of recommendations on food safety issues (see Box: *What the JPC said*). In July 2004, the BIS finalised draft standards for soft drinks, and in the same month, the Union health ministry issued a notification specifying that the quality of water used in making soft drinks should meet bottled water standards.

4. *Why was the second study done? What did it find?*

In August 2006, CSE conducted a follow-up study on soft drinks and found them to be still very unsafe. Worse, even the directions given by the JPC had been disregarded. The standards had been finalised, but blocked because of opposition from the industry.

The study tested 57 samples of 11 soft drink brands, from 25 different manufacturing plants of Coca-Cola and PepsiCo, spread over 12 states in India. It found a cocktail of an average of three-five

BOTTLED WATER CAME FIRST

In 2003, CSE analysed 17 brands of packaged drinking water sold in and around Delhi and 13 brands from the Mumbai region. The samples were found to contain a deadly cocktail of pesticide residues. Most of the samples contained as much as five different pesticide residues, in levels far exceeding the standards specified as safe for drinking water.

CSE had tested for two types of pesticides – organochlorine and organophosphorus: the most commonly found pesticides were lindane, DDT, malathion and chlorpyrifos. The samples had enough poison to cause, in the long term, cancer, liver and kidney damage, disorders of the nervous system, birth defects, and disruption of the immune system.

CSE used European norms because the standards set for pesticide residues by the Bureau of Indian Standards (BIS) were vague and undefined. CSE's campaign bore fruit when the government in July 2003 decided to notify new norms for pesticide residues in bottled water. The norms stated that pesticide residues considered individually should not be more than 0.0001 mg/litre while total pesticide residues were capped at not more than 0.0005 mg/litre.

WHAT THE JPC SAID

"The Committee... find that the CSE findings are correct on the presence of pesticide residues in carbonated water strictly in respect of the 36 samples of 12 brand names analysed by them. The Committee also appreciate the whistle blowing act of CSE in alerting the nation to an issue with major implications in food safety, policy formulation, regulatory framework and human and environmental health."

pesticide residues in all samples – 24 times higher than the BIS norms which had been finalised but not notified.

The levels in some samples such as Coca-Cola bought in Kolkata exceeded the BIS standard by 140 times for the deadly pesticide lindane. Another Coca-Cola sample from Thane contained the neurotoxin chlorpyrifos at 200 times the standard.

5. How did the two CSE studies compare?

Both the studies were conducted by CSE's Pollution Monitoring Lab. In 2003, the lab had been severely criticised by the soft drinks companies; however, the JPC had upheld its methodologies and findings. In 2006, the lab introduced some changes – it got an ISO 9001:2000 quality management system accreditation, and confirmed the presence of pesticides in the samples using an expensive and state-of-art equipment, the GS-MS.

Taking the yet to be notified BIS standards into consideration, the average level of pesticide residues in the samples was 34 times above the standard in 2003; and 24 times more in 2006. However, CSE found pesticide residues as high as 52 times the level in the samples bought in Kolkata, 42 times the level in the samples bought in Nainital and Gorakhpur, and 34 times the level in the samples bought in Mumbai (see Tables: *The two studies through numbers* and Box: *Some conclusions*).

6. How did the industry respond to the second CSE study?

Cola companies claimed their products met standards and were safe; Coca-Cola said there were "no detectable levels of pesticides" in its soft drinks, and that its products has been regularly tested and evaluated by a renowned UK-based lab (see Box: *The UK lab study*). However, the company failed to publish the test results to prove the safety of its products. Three years ago, in response to CSE's findings, soft drink companies had issued similar statements claiming they were meeting "strict European Union standards". But tests by several other labs, including renowned government labs like CFL, Kolkata, CFTRI, Mysore and CPCB, Delhi, had found that they did contain high levels of pesticide residues and were not meeting the norms as they claimed.

Ironically, these same companies repeatedly told the government that their products were a complex matrix and therefore could not be tested. The companies also released advertisements in newspapers and television, claiming their products were clean. In a shameful show of desperation, the companies threatened to stop the flow of foreign direct investment (FDI) into the country. They contended that a standard for a final product (the bottle of soft drink) could not be set. Instead, they

THE TWO STUDIES THROUGH NUMBERS

Brand	Total organochlorines (ppb)		Total organophosphates (ppb)		Total pesticides (ppb)	
	2003	2006	2003	2006	2003	2006
Coca Cola	4.4	6.97	17.9	6.41	22.3 (44.6)	13.38 (26.8)
Fanta	6	5.6	15.4	3.53	21.4 (42.8)	9.14 (18.3)
Limca	4.7	4.325	10.1	2.65	14.8 (19.6)	6.97 (14)
Thums-Up	1.5	7.588	9.6	3.35	11.1 (22.2)	10.93 (21.9)
Pepsi	3.2	9.1	15.5	6.11	18.7 (37.4)	15.21 (30.4)
7-Up	3.6	5.9	13	6.56	16.6 (33.2)	12.46 (24.9)
Mirinda orange	5	6.84	14.6	3.89	19.6 (39.2)	10.68 (21.4)
Mirinda lemon	8.4	7.12	26.8	0.96	35.2 (70.4)	8.08 (16.2)
Mountain Dew	3.3	3.6	10.8	3.37	14.1 (28.2)	6.97 (13.9)
Average	4.45	6.34	14.85	4.08	19.31 (38.6)	10.44 (20.9)

Notes: Figure in parenthesis indicates the number of times total pesticide count is higher than the standards of the European Union (0.5 ppb)

SOME CONCLUSIONS

- 2003: 36 samples of 12 different brands. Lindane, DDT and its metabolites, malathion and chlorpyrifos found.
- 2006: 57 samples of 11 different brands. Lindane, chlorpyrifos, heptachlor and malathion found.
- Total pesticide content of most samples lower in 2006, mainly because of the reduction in the organophosphorus pesticides' content.
- Average total pesticides content: 19.08 ppb in 2003, 10.76 ppb in 2006. This was, however, still much higher than the EU norm of 0.5 ppb.
- Organochlorine pesticide content: Higher in 2006. Except Fanta, all brands had more organochlorine pesticides. Average content went up to 6.3275 ppb in 2006 compared to 4.4556 ppb in 2003.
- Lindane: present in all samples in both years. Average concentration went up from 2.1 ppb in 2003 to 5.37 ppb in 2006 (much higher than the prescribed EU limit of 0.1 ppb)
- DDT: Detected in 81 per cent of samples in 2003; absent in 2006.
- Chlorpyrifos: Present in all samples in both years. Concentration rose to 4.71 ppb from 4.2 ppb.
- Malathion: Present in 97 per cent of samples tested in 2003 (average concentration of 8.7 ppb) and 39 per cent of samples tested in 2006 (average concentration of 0.17 ppb).
- Heptachlor: Organochlorine pesticide banned in India, detected in 72 per cent of the samples at an average concentration of 0.41 ppb, which is 4.1 times the BIS limit.

THE UK LAB STUDY

In 2006, London-based Central Science Laboratories (CSL) tested one bottle of Coca-Cola and gave the product a clean chit. The laboratory's limit of quantification (LOQ) was 0.5 to 2.5 ppb – it could only detect and quantify pesticide levels in soft drinks if these levels were 5-25 times higher than the EU limit (or the BIS's final but not notified standard). Implying, it could not detect pesticides because its equipment did not have the sensitivity to find them. In comparison, the CSE lab's LOQ is 0.01-0.2 ppb.

There were other glitches: to begin with, the samples were provided by Coca-Cola itself. The lab had not collected them independently and randomly from the open market – as was done by CSE for its study. How could the UK use an industry sponsored and funded study for regulatory purposes?

Secondly, the data provided by Coca-Cola were for samples tested in June 2005. Then why was the report being released a year later? Was the laboratory not testing samples regularly?

Thirdly, the laboratory used the name of the UK government's department for environment, food and rural affairs. But will the UK government allow contamination beyond stipulated levels in its own country? Around the time the study was done, the UK government recalled beverages from the market when these were found to exceed the non-existent domestic benzene standards by just 1 ppb. In this case, the government used WHO's (World Health Organization) drinking water standards to say that these products were unsafe and must be recalled. In our case, we have found soft drinks to exceed the final (but not notified) standards by 10-50 times. Would the UK government have allowed this? Or is our life cheaper?

Fourthly, the laboratory said it did not find any pesticide residues. What was the laboratory's limit of detection? Laboratories can only check above their limits of detection and quantification, which specify the sensitivity of their equipment.

wanted the government to regulate their inputs – agricultural raw commodities and water: implying they only wanted standards for the water and sugar they use, not the product that they sell.

CSE contested the companies' claims – if they claimed their products were too complex to test, how was the UK lab testing them? Were they saying that only a Western lab could test their products? CSE used the methodology laid down by the USEPA and used by governments across the world for testing liquid matrices – any complex multi-ingredient product which has water and other substances.

7. *What support did the industry find from the government?*

Following the release of the UK lab report, the then Union minister of health and family welfare, Anbumani Ramadoss, gave a statement in the Parliament questioning the validity of CSE's findings. The minister decided to toe the company line and even used its language *verbatim*, exposing his allegiance to the industry.

And this wasn't the first time that the government was siding with the companies. In 2003, when CSE released its study on soft drinks, the then government had issued a statement in Parliament

saying that the companies were within safety limits. This 'clean chit' was used by the companies to proclaim the safety of their products.

8. Did CSE face any threats from the industry?

On August 25, 2006 the pesticide industry threatened CSE with legal action for its campaign against pesticide residues in soft drinks. CSE was not the only institution which was at the receiving end of the industry's rant: other experts, scientists and institutions received equal attention.

CSE maintains that the only purpose of these threats was to "criminally intimidate people and organisations who work on public health by threatening with malicious prosecution. This practice is completely wrong and stifles research, free speech and public voice by threatening people with unfounded legal notices and prosecution".

Such lawsuits, where the rights of individuals or institutions to bring matters of public interest to the notice of the public are questioned, are common in countries like the US; common enough to be given a

POINT COUNTER-POINT

The questions raised by the health ministry on the CSE study based on the UK lab report:

a) What the government said: Heptachlor is banned since 1996, so its presence is unlikely.
CSE's rebuttal: Heptachlor was banned because it is a highly persistent pesticide. Once used, it is likely to be found in the environment for more than 20 years. Therefore, not only will heptachlor be found today; it will continue to be found for the next 10 years. The very scientists in the ministry's expert group, who were questioning heptachlor findings, had themselves found this pesticide in different kinds of samples – water, vegetables, milk – from all over the country. GC-MS chromatograms in CSE's detailed report indisputably confirms heptachlor presence in the samples tested by us.

b) What the government said: The prevalence of delta HCH is in contradiction to its biological nature.
CSE's rebuttal: Government's own labs, and various research organisations all over the country, have also found this "contradiction". Residues of delta HCH have been found in numerous samples of milk, drinking water, pond water, vegetables, soil samples and even in rainwater all over the country. Delta HCH has been found in samples from Bathinda, Thiruvananthapuram, Hissar, Sirsa and Rohtak, Delhi, Shahjahanpur, Haridwar and other parts of India.

If CSE's findings are "contradictory" then so are those of such eminent government organizations as the Indian Council of Medical Research, the Indian Council of Agricultural Research and the Chandigarh-based Post Graduate Institute of Medical Education and Research.

c) What the government said: The CSE report does not provide details required for the confirmatory interpretation of quantum results.
CSE's rebuttal: The report put on our website and scrutinised by the ministry's expert committee is a scientifically standardised report, following universally accepted norms. Our complete investigation of cola samples is compiled as a 452-page report, comprising GC-ECD/NPD chromatograms and GC-MS spectra. The full report is available for public scrutiny.

d) What the government said: Malathion residue is technically unlikely and the GC-MS also confirms absence of malathion.
CSE's rebuttal: The same issue was raised and analysed in detail by the JPC in 2003-04. CSE's malathion findings withstood scientific scrutiny and were endorsed by the JPC. In the 2006 study, CSE double checked its malathion findings and confirmed it through its GC-MS equipment. The GC-MS report unequivocally detects and confirms malathion. This is the most credible, and scientifically advanced, evidence to show the presence of malathion.

e) What the government said: Chlorpyrifos is present but the retention time does not match.
CSE's rebuttal: We thank the experts for detecting this dangerous pesticide in colas. However, so far as the 'retention time' is concerned, it is a general scientific understanding that the retention time of a pure compound such as chlorpyrifos in GC-MS and that of the same compound in a food commodity will always vary, because the food commodity also contains chemicals other than that compound. In GC-MS analysis, the retention time is not as important. as the identification of the compound by the GC-MS.

f) What the government said: The literature review of the CSE report is not balanced.
CSE's rebuttal: The literature review talks about issues other than pertaining to the test report. In our review, we have talked about constituents of soft drinks such as caffeine, acids and other additives. All these constituents are harmful. Thus, the ministry's criticism itself is unbalanced.

name: Strategic Lawsuits Against Public Participation or SLAPP for short. SLAPPs amount to silencing people into submission. They are not just "intimidation lawsuits", but question the rights of individuals and institutions to speak out on a public issue, to communicate their views to government officials. They question the right of people to tell their elected representatives what they think, want, or believe in – in effect, for attempting to influence government action.

9. Why should standards be set for safe levels of pesticide residues in soft drinks?

CSE's two studies established conclusively that bottles of soft drinks – carbonated beverages – did harbour a host of pesticide residues in them. Companies retorted with the argument that these soft drinks were still safe, because the pesticide residues detected in them were in tiny quantities and the levels were lower than what is found in other products such as milk or juices.

A TIMELINE

- August 6, 2003: Issue discussed in Lok Sabha; members express serious concern about the findings on pesticide residues in soft drinks and ask government to explain. Soft drinks are banned from Parliament premises. Sushma Swaraj, the then health minister, orders testing of soft drink samples at government labs – the CFTRI in Mysore and CFL in Kolkata.
- August 21, 2003: Swaraj presents the results of tests conducted by the government labs. Gives a statement on the matter in Parliament: admits the tests did find pesticide residues, but says the levels were "well within the safety limits as per existing standards of packaged drinking water". However, ignores something the reports clearly state – that their findings were not comparable to CSE findings as samples were different in both cases. MPs demand setting up of a Joint Parliamentary Committee (JPC) to look into the issue.
- August 22, 2003: Government announces JPC, chaired by Sharad Pawar.
- August 26, 2003: The Union government issues a draft amendment notification of the PFA, 1955 to regulate beverages and other food products. Clubs all beverages into one category and stipulates stringent standards for all.
- January 27, 2004: The JPC adopts its report unanimously.
- February 4, 2004: JPC report is tabled in Parliament. Upholds the CSE findings. Whistle-blowing act of CSE appreciated. Makes a wide range of recommendations on food safety issues.
- February 13, 2004: Emergency meeting of the Central Committee on Food Standards (CCFS) called to discuss the JPC report. Report endorsed; committee says it will finalise standards for soft drinks urgently.
- June 23, 2004: The Pesticide Residues Sub-Committee of the CCFS meets to finalise standards. However, committee recommends year-long monitoring of pesticide residues in soft drinks, before standards are fixed.
- July 15, 2004: BIS finalises draft standards for soft drinks, based on JPC recommendations; standards for the final product, include pesticide residue standards and global best standards for caffeine. The draft standards are available for public comment till August 31, 2004. If accepted, these would be the world's first standards for pesticide residues in soft drinks.
- July 27, 2004: The Union ministry of health and family welfare issues a notification specifying that the quality of water, which will be used in the manufacture of soft drinks, must meet the bottled water standards.
- March 29, 2006: In an effort to scuttle the efforts of the BIS committee which was finalising the standards, a letter by the then health secretary asks the BIS not to finalise the standards, using the pretext of constituting more committees.
- August 2, 2006: CSE releases it second study on soft drinks and finds they continue to be unsafe.
- August 2006: Kerala imposes a ban on the sale of soft drinks.
- August 25, 2006: The pesticide industry threatens CSE with legal action for its campaign against pesticide residues in soft drinks.
- March 13, 2007: Ganguly Committee files its report in the Supreme Court; confirms the presence of pesticides in soft drinks and paves the way for the health ministry to finalise and notify the standards for carbonated beverages.
- August 17, 2008: The Union health ministry notifies the rules for maximum levels of pesticides in carbonated water. Sets rules for eight pesticides.

This is nothing but scientific jugglery. "Safety is not only about high or low numbers," explains Sunita Narain, director general, CSE. Pesticides are tiny toxins and deadly for us if we are exposed to quantities higher than what is defined as an acceptable limit. In other words, our exposure to these toxins, through the food we eat and water we drink, must be kept under the threshold of safety. The safe limit – or the standard – in each product is set keeping in mind the nutrition-pesticide trade-off: a quota for residues can be allowed in nutritive food like milk or juices, but not in non-nutritive products like carbonated beverages. This is why CSE demanded that the government must set standards for safe levels of pesticide residues in soft drinks.

10. *What is the current state of affairs in this standard-setting game?*

The draft standards created by BIS have set a limit for 16 pesticides in soft drinks. The limit of individual pesticide residues has been fixed at 0.0001 mg/l, and the total pesticide residues are not to exceed 0.0005 mg/l. The pH of the carbonated beverage has been set to be not less than 2.3. The caffeine content has been scaled down from 200 mg/l to 145 mg/l. Manufacturers will have to follow the labeling standards. Anything beyond the latter would have to be labeled as "is not recommended for children, pregnant or lactating women, and individuals sensitive to caffeine".

However, the Union ministry of health and family welfare, egged on by industry, had effectively blocked the standards for carbonated beverages. This is evident from a confidential letter written by the then health secretary on March 29, 2006 to the BIS committee which was finalising the standards. The letter asked the BIS not to finalise the standards, using the pretext of constituting more committees. This was clearly a prevarication, which suited the companies' interests.

In April 2007, a committee headed by N K Ganguly, director general, Indian Council of Medical Research, made its recommendations, mooting a final product standard to regulate soft drinks (see Box: *The Ganguly committee*). The Ganguly committee, also known as the National Level Expert Group, was constituted in November 2004 by the health ministry to guide the authorities in fixing the "maximum residue level limits of pesticides in carbonated beverages, fruits and vegetable juices and other finished products" and "on monitoring of pesticides in carbonated beverages", and "based on the contents of the soft drinks/beverages to advise on their harmful effects".

The committee's report, filed in the Supreme Court on March 13, 2007, paved the way for the Union ministry of health and family welfare to finalise and notify a mandatory final product standard for pesticide residues in soft drinks.

On August 17, 2008, more than five years after the first CSE study, the ministry notified the draft rules for maximum levels of pesticides in carbonated

THE GANGULY COMMITTEE

- Recommended a maximum residue level of 1 ppb for an individual pesticide for carbonated water.
- Recommendation based on a public health risk assessment of soft drinks built on an estimation of quantities consumed.
- The committee tested samples of sugar, sugar syrup and soft drinks, and found no pesticide residues in the sugar; companies had stridently argued that Indian sugar was contaminated.
- Made it clear that colas can be reliably tested – something which the cola companies had claimed could not be done.
- Flaw: Even the limits prescribed for individual pesticides are 10 times the 0.1 ppb limit finalised by the BIS
- Flaw: No limit prescribed for total pesticide residues.

water. Under a proposed amendment to the then Prevention of Food Adulteration (PFA) Rules (now replaced by the Food Safety and Standards Act, 2006), the ministry specified 1 ppb (parts per billion) as the maximum permissible limit for lindane, DDT, malathion and five other pesticides.

It is noteworthy that this limit is 10 times higher than what had been finalised for packaged drinking water in 2003 – 0.1 ppb for residues of individual pesticides and 0.5 ppb for that of total pesticides.

India has mandatory rules under the Food Safety and Standards Act, 2006. The BIS has draft standards which are more stringent – 0.1 ppb for individual pesticides and 0.5 ppb for total pesticide residues. The soft drink industry is following the less stringent mandatory norms.

11. What is CSE asking for?

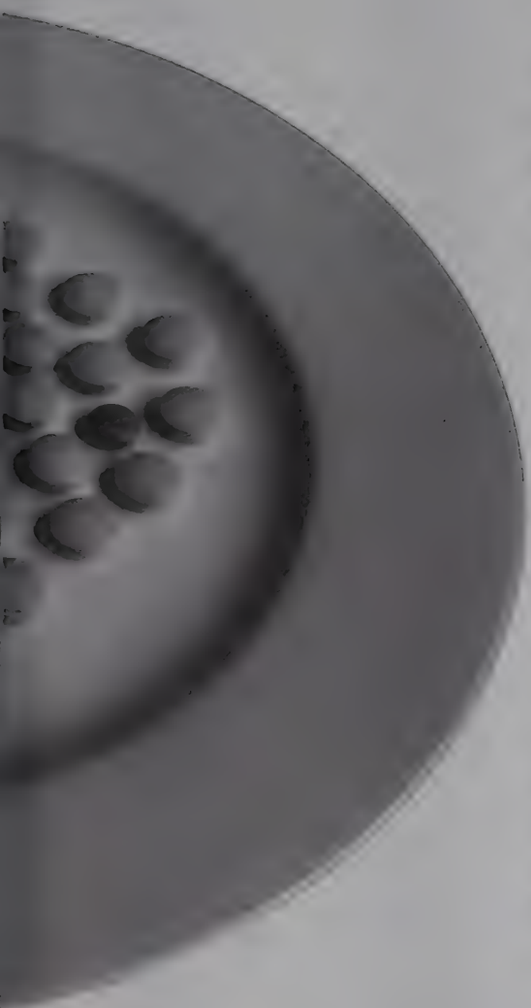
CSE demands that the stringent (and currently voluntary) BIS norms must be made mandatory so that the industry is forced to follow them. Soft drinks, consumed particularly by children, need to be checked and regulated.

CSE also thinks there is no need for any further studies, reviews or committees. The BIS committee had, since 2003, addressed each and every issue raised by the cola companies and, based on sound science, has finalised the standards. ●

ANTIBIOTICS IN FOOD

2

Antibiotics in our food are now a global concern because of their adverse health impacts and for proliferating antibiotic resistance in bacteria, making antibacterial treatment ineffective. The World Health Organizations says antibiotic resistance is "one of the three greatest threats to human health". In India, antibiotics are used widely in food animals as growth promoters and to prevent and treat infections. However, there is no data on this usage; neither is there any regulatory provision regarding the use of antibiotics in livestock. In India, only aquaculture has rules on antibiotics, but these are flouted.



In 2002, EU inspectors ordered the destruction of three large consignments of shrimp from India after chloramphenicol was detected. Chloramphenicol is banned in India for use in aquaculture.[1] In 2009, within a period of six months, the EU rejected more than 50 shrimp consignments from India due to detection of an antibiotic residue, presence of bacteria, traces of heavy metals and unhygienic condition of the products. The total number of rejections in 2008 was around 30.[2]

Not only poultry and livestock, even natural products like honey that people consume because of their rejuvenative and immunity-boosting properties, are now contaminated with antibiotics. The EU has also banned Indian honey on account of presence of antibiotics and heavy metal residues.

In 2009-10, CSE tested honey sold in Indian markets and found that 11 out of the 12 samples tested contained high levels of antibiotics. Not only did the study show that what we consider natural and pure was contaminated, but also that there were very few regulations for antibiotics in food and no regulation for antibiotics in honey in India.

2.1 HONEY

SAYANTONI PALCHOUDHURI / CSE

1. Why did CSE test for antibiotics in honey?

In June 2010, the European Union (EU) had banned Indian honey; EU had found it contaminated with high amounts of antibiotics. To prevent further damage to the export of Indian honey, an elaborate system of monitoring was put in place. The Exports Inspection Council (EIC), entrusted with the task of checking exports, set antibiotic standards for honey which is exported.

These standards, however, do not apply to the honey sold in the domestic market, or the honey which India imports. Having come up against this regulatory black hole, CSE's Pollution Monitoring Laboratory (PML) decided to probe how much antibiotic-laden is the honey sold in the domestic market.

2. What were the key findings of the CSE study?

CSE tested 12 samples of honey, of which two were imported (see Box: *What the CSE study was all about*). Multiple antibiotics in high amounts were found in 11 out of the 12 samples. All 11 samples failed the antibiotics standards set by the Indian government's Export Inspection Council (EIC) for exported honey.

These findings provided some key insights: firstly, that the honey industry rampantly uses antibiotics. Also, the fact that more than one antibiotic was found in the samples indicates that the practice is to collect honey from different sources and then blend them before packing and distributing for sale. So one does not know from where the honey has been sourced. This lack of traceability calls for a need to regulate and monitor the level of antibiotics in honey being sold in the domestic market, as continuous long-term exposure to low levels of antibiotics could in due course lead to antibiotic resistance in pathogenic bacteria, making their treatment difficult.

The domestic branded honey market is estimated at around Rs 250 crore. Dabur India Limited is the biggest player here, with a share of over 75 per cent. The remaining share is distributed between brands like Baidyanath, Himani, Zandu, Mehsons, Himalaya and other smaller companies. Almost all the samples of these and other brands were found contaminated with antibiotics; the sole exception was Hitkari Honey of Hitkari Pharmacy, Delhi.

HONEY - WHAT THE CSE STUDY WAS ALL ABOUT

Honey is a natural product, and can be used as an instant energiser as it contains sugars which are quickly absorbed by our digestive system and converted into energy; it also has antioxidant and antibacterial properties. Used with other herbal preparations, honey can enhance the medicinal qualities and reach of these preparations. The quality, taste, texture, color and aroma of honey differs according to the geographical area as well as the plants from which the bees collect it (for instance, honey gathered from eucalyptus plantations would differ from that taken from mustard farms).

THE METHODOLOGY

The samples were analysed in triplicate (each sample was tested thrice and the results given as average of three tests) using High Performance Liquid Chromatography (HPLC) with Diode Array Detector (DAD) and Fluorescence Detector (FLD). Internationally accepted published methods were used for analysis and validated by the laboratory. The results were confirmed by spiking.

THE SAMPLE

The 12 honey samples were bought randomly by the CSE lab from various markets in Delhi in July 2009. Of these, 10 were Indian and two were imported brands. None of the packages mentioned the source of the honey; it is, therefore, difficult to say whether the honey was produced and packaged in India, or was imported and then packaged in India, or was a mix of imported and domestic honey.

THE DOMESTIC BRANDS INCLUDED:

- Dabur Honey of Dabur India Ltd, which holds over 75 per cent of the market share in the branded segment.
- Himalaya Forest Honey of Himalaya Drug Company, one of India's oldest Ayurveda drug companies.
- Patanjali Pure Honey of Patanjali Ayurved Ltd, Haridwar.
- Baidyanath Wild Flower Honey of Shree Baidyanath Ayurved Bhavan Pvt Ltd (Kolkata), a well known name which has about 10 per cent share in the branded honey market.
- Khadi Honey of Khadi Gramodyog Sewa Samiti, Bihar.
- Mehsons Honey of Mehsons India Ltd, a Bareilly-based company involved in the honey and beeswax business; it also exports medicines and herbs.
- Gold Honey of Vardhman Food & Pharmaceuticals, Faridabad, Haryana.
- Umang Honey of Udyog Bhartiya, Delhi, a registered Khadi and Village Industries Society.
- Hitkari Honey of Hitkari Pharmacy, Delhi.
- Himflora Gold of Food Max, Noida.

THE TWO IMPORTED BRANDS WERE:

- Capilano Pure & Natural Honey of Capilano Honey Ltd, Australia. This company is the market leader in Australia, exports honey to over 40 countries, and has large markets in Asia, North America, Middle East and Europe.
- Nectaflor Natural Blossom Honey of Narimpex AG with its production site in Biel, Switzerland. It is a well-known supplier of honey, dried fruits and nuts.

THE RESULTS

- Multiple antibiotics in high amounts were found in 11 out of the 12 samples. All 11 samples failed the antibiotics standards set by the Indian government's Export Inspection Council (EIC) for exported honey.
- The two imported honey samples were also highly contaminated with antibiotics. Both would have failed their own domestic standards.
- The only sample in which no antibiotic residues were detected was Hitkari Honey of Hitkari Pharmacy, Delhi. This is a small company involved in the seasonal honey business.
- The fact that more than one antibiotic was found in the samples indicates that the practice is to collect honey from different sources and then blend them before packing and distributing for sale. So one does not know from where the honey has been sourced.
- There is a need to regulate and monitor the level of antibiotics in honey being sold in the domestic market, as continuous long-term exposure to low levels of antibiotics could in due course lead to antibiotic resistance in pathogenic bacteria, making their treatment difficult.

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ANTIBIOTICS FOUND

Antibiotic	Details of the antibiotic	Samples/brands in which it was found	Quantity/ concentration	Comparison of level/quantity to standard	Health impacts
Oxytetracycline	Belongs to the common antibiotic class of tetracyclines. Is used by beekeepers against the bacterial foul brood disease	Found in 50 per cent of the samples (6 out of 12)	Ranging from 27.1-250.4 µg per kg	Almost 3-25 times higher than the 10 µg/kg EIC standard	Chronic exposure can lead to blood-related disorders, liver injury and blood coagulation; can damage calcium rich organs like teeth; may cause eroded nasal cavities, sensitivity to the sun, wheezing and asthmatic attacks; children under seven years may develop a discoloration of the teeth
Chloramphenicol	Banned from use on food-producing animals in many countries	Found in 25 per cent of the samples (3 out of 12)	Found in the range of 3.6-4.4 µg/kg	12-15 times higher than the 0.3 µg/kg EIC standard	Potential carcinogen and genotoxin; repeated or prolonged exposure can lead to organ damage, bone marrow toxicity, and aplastic anemia; increases the risk of childhood leukemia
Ampicillin	Widely used in veterinary medicine for treatment and prevention of bacterial diseases; not recommended for use on honeybees	Found in 67 per cent of the samples (8 out of 12)	Concentrations of 10.1-614.2 µg/kg	There are no standards for ampicillin in honey in any country, as it is not supposed to be used in beekeeping. Ampicillin, therefore, is an unauthorised and illegal substance in honey	Repeated exposure may cause allergic reactions, asthmatic attacks etc
Enrofloxacin	Belongs to the fluoroquinolone class. Used to treat bacterial infections and as a growth promoter in cattle; reportedly, beekeepers are using it as well	Found in 10 out of 12 samples (83 per cent)	Ranging between 10.9-144.8 µg/kg	No standard. Illegally present	Could be a terratogen, lethal for embryos; the US Food and Drug Administration has proposed to withdraw approval for the use of fluoroquinolone antibiotics in poultry because of the drug's potential for increasing human pathogen resistance
Ciprofloxacin	Metabolite of enrofloxacin; used in poultry farming	Found only in Baidyanath Wild Flower Honey	19.9 µg/kg	No standard. Illegally present	Same as above
Erythromycin	Used for poultry; now being used to protect honeybees from bacterial diseases	Found in 42 per cent of the samples (5 out of 12)	Ranging between 69.7-280.3 µg/kg	No standard. Illegally present	Chronic exposure can turn it into a terratogen; can lead to cardiac malfunctions in infants of women administered this antibiotic during pregnancy; exposure to it linked to increased probability of pyloric stenosis (a condition affecting the gastro-intestinal tract, leading to increased vomiting) in infants.

Notes: µg - microgram; kg - kilogram

THE BRANDS AND HOW THEY FARED

Brand	Antibiotics present	Quantity present	Comparison with EIC standard
Dabur Honey	3 out of the 6 antibiotics tested found in this brand – oxytetracycline (OTC), ampicillin and enrofloxacin	91.3 µg/kg of OTC, 26.6 µg/kg of ampicillin and 88.7 µg/kg of enrofloxacin	Level of OTC is 9 times the EIC standard
Himalaya Forest Honey	3 antibiotics detected – ampicillin, enrofloxacin and erythromycin	23.8 µg/kg of ampicillin, 63.8 µg/kg of enrofloxacin and 69.7 µg/kg of erythromycin	Sample non-compliant, as there are no standards
Mehsons Pure Honey	2 antibiotics detected – enrofloxacin and erythromycin	58.3 µg/kg of enrofloxacin residues and 85 µg/kg of erythromycin	Sample non-compliant
Himflora Gold Honey	2 antibiotics detected – enrofloxacin and ampicillin	35.5 µg/kg of ampicillin and 37.7 µg/kg of enrofloxacin	
Patanjali Pure Honey	4 antibiotics detected – erythromycin, OTC and ampicillin	27.2 µg/kg of OTC, 30.5 µg/kg of ampicillin, 75.17 µg/kg of enrofloxacin and 186 µg/kg of erythromycin	OTC almost 3 times the EIC standard
Baidyanath Wild Flower Honey	2 antibiotics detected – ciprofloxacin and ampicillin	19.9 µg/kg of ciprofloxacin and ampicillin at a level of 25.2 µg/kg	Sample non-compliant, as there are no standards
Khadi Honey	3 antibiotics detected – enrofloxacin, OTC and ampicillin	250.4 µg/kg of OTC, 10.1 µg/kg of ampicillin and 10.9 µg/kg of enrofloxacin	OTC level is 25 times the EIC standard – highest level among all the pesticides tested
Gold Honey	4 antibiotics detected – OTC, chloramphenicol, enrofloxacin and erythromycin	57.7 µg/kg of OTC, 4.4 µg/kg of chloramphenicol, 34.3 µg/kg of enrofloxacin and 231.3 µg/kg of erythromycin.	OTC almost 6 times the EIC standard, while chloramphenicol at its highest among all the tested brands (15 times higher than the EIC standard)
Hitkari Honey	No antibiotics detected		
Umang Honey	2 antibiotics detected – ampicillin and enrofloxacin	208.1 µg/kg of ampicillin and 122.1 µg/kg of enrofloxacin	Sample non-compliant with EIC standards
Capilano Pure and Natural Honey (Australia)	3 antibiotics detected – OTC, chloramphenicol and enrofloxacin	150.8 µg/kg of OTC, 3.6 µg/kg of chloramphenicol, 144.8 µg/kg of enrofloxacin	OTC 15 times the EIC standard, but within the Australian standard of 300 µg/kg; chloramphenicol, which is banned for food production in Australia, 12 times over the EIC standard
Nectaflor Natural Blossom Honey	Highest number detected in this brand – 5 antibiotics: OTC, chloramphenicol, ampicillin, erythromycin and enrofloxacin	112.0 µg/kg of OTC, 3.6 µg/kg of chloramphenicol, 614.2 µg/kg of ampicillin, 280.3 µg/kg of erythromycin, 56.1 µg/kg of enrofloxacin	OTC 11 times the EIC standard; chloramphenicol, banned by the EU, 12 times over the EIC standard

Notes: µg - microgram; kg - kilogram
Source for both the tables: *Down To Earth*, September 16-30, 2010

3. What are the international standards for antibiotics in honey?

The Codex Alimentarius Commission is a global body set up jointly by the UN's Food and Agriculture Organization (FAO) and the World Health Organization (WHO) to develop food standards for international trade. Since honey is an internationally traded commodity, the Codex has set down standards for the quality of the honey that is traded. However, it has nothing to say on the presence of antibiotics in honey.

The European Union (EU) regulates honey under the Council Directive 2001/110/EC. The standard for antibiotics in food (referred to as Maximum Residue Limits or MRLs) stipulates that each antibiotic must have an MRL before it can be used on a food-producing species. But there are no MRLs for antibiotics in honey, which means EU does not allow the use of antibiotics for treatment of honeybees.

For regulating residues of antibiotics in imported honey, the EU has set up 'Reference Points for Action' or RPAs. RPAs are residue concentrations, which are technically feasible to detect by food control laboratories. When a laboratory detects

antibiotics, the consignment is rejected. Till date, RPAs have been established in honey for substances such as chloramphenicol and nitrofuran. The EU has also set a provisional MRL of 25 parts per billion (ppb) for oxytetracycline in honey.

In the US, MRLs for antibiotics in food are set by the US Food and Drug Administration (US FDA), and listed in Title 21, Part 556 (21 CFR 556). There are no limits for antibiotics in honey (see Table: *No standards*). Regulators in the EU and US say that antibiotics are 'unauthorised' and therefore 'illegal', unless there is a standard regulating their levels. This is the reason why the EU banned Indian honey from entering after it was found contaminated with high amounts of antibiotics.

4. What are our national standards regulating antibiotics in honey?

Honey is promoted and monitored by multiple agencies for exports, but its imports and domestic consumption are managed by the Food Safety and Standards Authority of India (FSSAI) – and it has no rules on antibiotics in honey. In fact, the CSE study set the ball rolling and the government has begun the process of making rules. The Food Safety and Standards Act, that replaced the Prevention of Food Adulteration (PFA) Rules, 1955, regulates honey. The Act has now added a clause that no antibiotics will be allowed in honey, and the FSSAI is framing rules for testing of antibiotics in honey.

The Bureau of Indian Standards (BIS) has a norm for extracted honey under IS 4941:1994. Brands wishing to obtain the ISI mark (also known as Standard Mark that suggests conformity to BIS rules) have to follow it. Under it, honey is classified into three grades based on the moisture content. There is also the Honey Grading and Marking Rules, 2008 under the Agricultural Produce (Grading and Marking) Act, 1937 (AGMARK) which is implemented by the Union ministry of agriculture. However, neither the BIS nor the AGMARK Rules have yet prescribed a standard for antibiotics in honey.

Besides these, there are a number of other agencies that are responsible for marketing and promoting the product. The National Bee Board, under the National Horticulture Mission, is mandated to promote best practices. All beekeepers ought to be registered with the board to ensure honey can be traced from the fork to the farm; however, very few are registered.

The Agriculture and Processed Food Products Export Development Agency (APEDA), under the Union ministry of commerce, provides financial assistance and subsidies to honey exporters. Between 2005 and 2008, the agency was monitoring and testing honey for exports. The Export Inspection Council (EIC), another department of the commerce ministry, certifies safety of food products, including honey. It is recognised by the EU. The council conducts tests on honey samples before they leave for Europe and is responsible for year-long monitoring of both exporters and beekeepers.

5. What is the source of antibiotics in honey?

Antibiotic traces in honey come from the bees that are treated with antibiotics. Beekeepers largely use these antibiotics to cure bacterial diseases, but some also use them to promote growth and production (see Box: *How antibiotics in honey came to India*).

Today, honey is produced in an environment polluted by different sources of contamination. Environmental sources of this contamination include pesticides, heavy metals, bacteria and radioactivity. Contamination also comes from beekeeping practices (apiculture): acaricides used to control parasitic mites, bee repellants used during harvesting season, pesticides for wax moth, etc. Antibiotics like oxytetracycline, for instance, are used

NO STANDARDS

Class	Antibiotic	Codex Alimentarius	EU	USA	EIC
Tetracycline	Oxytetracycline	No MRL	Provisional MRL: 25 ppb	No MRL	10 ppb
Amphenicol	Chloramphenicol	No MRL	No MRL RPA: 0.3 ppb	No MRL	0.3 ppb
Macrolide	Erythromycin	No MRL	No MRL	No MRL	No LOA
Beta-lactam	Ampicillin	No MRL	No MRL	No MRL	No LOA
Fluoroquinolones	Enrofloxacin	No MRL	No MRL	No MRL	No LOA
Fluoroquinolones	Ciprofloxacin	No MRL	No MRL	No MRL	No LOA

Notes: MRL = maximum residue level; ppb = parts per billion; RPA = Reference Points for Action; LOA = Level of Action
Source: *Down To Earth*, September 16-30, 2010

HOW ANTIBIOTICS IN HONEY CAME TO INDIA

In India, the use of antibiotics was triggered by the change in the profile of honeybees after the introduction of the Italian bee species Apis mellifera in 1965 by the Punjab Agriculture University.

Till then, honey had been sourced from bee colonies of native species like the Indian rock bee (Apis dorsata), Indian honeybee (Apis cerana), and the dwarf honeybee (Apis florea). The new bee species soon replaced the native bees in the apiaries of Jammu and Kashmir, Himachal Pradesh, Punjab, Haryana and Uttar Pradesh, as the honey yield from their hives was higher. Apis cerana produces four to five kg honey in a year as compared to 20 to 25 kg by Apis mellifera. Apis dorsata gives a yield of up to 40 kg but its honey is not considered good enough for exports.

With the change in the bee species, their disease profile also changed. Twenty-five per cent of the Apis mellifera colonies in India perished after a severe attack of Varroa mite in 2005. The mites were reportedly brought to India by infected queen bees from Nepal. The Italian bees are said to be docile and have poor defense. The bees suffered from the European foul brood disease around the same time; it was first detected in Mahabaleshwar in Maharashtra.

The Thai sac brood disease affected the Apis cerana, an otherwise hardy species, in the 1990s, which spread all along the Western Ghats. According to the beekeeping directorate of the Khadi Village Industries, between 1995 and 2010, about 93,000 bee colonies in Maharashtra perished due to the Thai sac brood disease.

India recommends only oxytetracycline for use in beekeeping – that too by a handful of universities. But as antibiotics are freely available in the market, there is no control and regulation on what beekeepers purchase and use. In fact, the National Research Project on Honeybee and Pollination does not recommend any antibiotic or pesticide.

The situation has aggravated as 90 per cent of the antibiotics used for both humans and animals are similar in composition: the only difference is the dosage. Entomologists recommend oxalic and formic acid solutions to get rid of Varroa mite and European foul brood disease. The broad-spectrum antibiotic oxytetracycline is recommended only in case of a severe attack of European foul brood disease. The antibiotic is mixed with sugar and left on the bee boxes for the bees to feed. It, however, should be avoided during the honey flow season, so that the antibiotic residues do not get into the honey.

SAYANTAN BERA / CSE

But there are others who use antibiotics like oxytetracycline to induce the queen bee to lay more eggs. And this many beekeepers attribute to the antibiotic packet, which shows a hen with many eggs!

to treat diseases such as the European foulbrood disease (caused by *Paenibacillus* larvae).

Reports are now emerging of tetracycline resistance in bacteria because of widespread use of the antibiotic. Other antibiotics such as erythromycin, lincomycin, monensin, streptomycin, enrofloxacin etc are also in use.

6. Why are antibiotics used in other foods?

Antibiotics are used both to accelerate growth in farm animals and for treating diseases. Typically, the kind of antibiotics used in farm animals are the same as or closely related to those used in humans. Either way, the excessive unregulated use of antibiotics has led to a rise in cases of antibiotic resistance amongst

INTERNATIONAL TRADE IN HONEY: THE CHINESE CONNECTION

The problem started with Chinese honey. China is the biggest producer and exporter of honey. In 2001, it produced 254,359 tonne of honey, three times the amount produced by the two other top rankers, the US and Argentina. China also led in exports, commanding a 29 per cent share. Most of this honey went to the US and EU. In the honey trade, there are two distinct groups of countries: consumer countries like the EU and the US and others like China, Argentina and Turkey that produce close to half the world's share of honey.

In 2001, the US consumed nearly 185,000 tonne of honey but produced just half the amount; Germany consumed about four times of what it produced. On the other hand, China and Argentina have little domestic consumption and export most of what they produce.

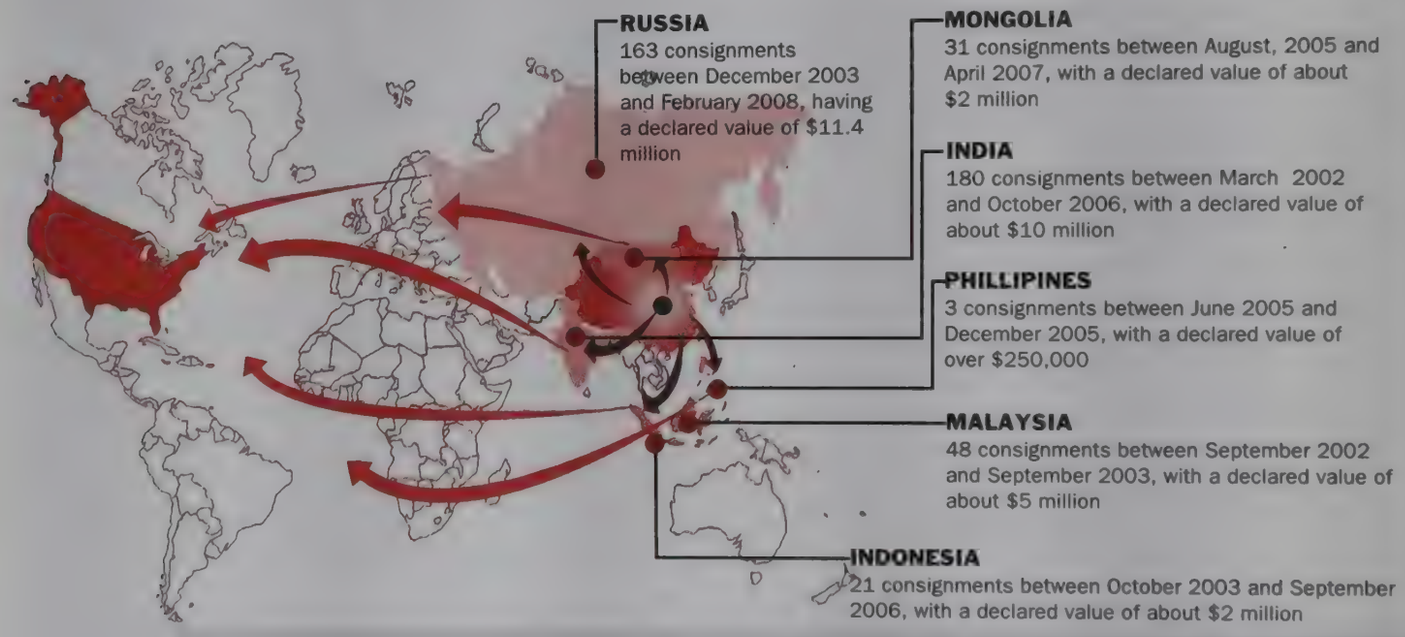
Honey Producers Association, which said the Chinese were dumping cheap honey in the US. The Chinese were accused of undercutting honey prices by about 150 per cent, which led to the US Department of Commerce slapping an anti-dumping duty of 221 per cent in 2001.

Export figures from Food and Agriculture Organization showed that Chinese exports fell because of the EU ban and the US duties, from 106,868 tonne in 2001 to 76,678 tonne the following year. This set the stage for illegal shipment of honey. This included re-routing thousands of tonnes of honey through countries like Russia, Indonesia, Thailand, Malaysia, Mongolia, Philippines and even India.

Chinese exporters changed labels on the consignments to show a different country of origin

CHINA TO US, VIA QUICK-CHANGE PORTS

ALW FOOD GROUP re-routed contaminated honey consignments from China through third countries to dodge US customs


RUSSIA
163 consignments between December 2003 and February 2008, having a declared value of $11.4 million
MONGOLIA
31 consignments between August, 2005 and April 2007, with a declared value of about $2 million
INDIA
180 consignments between March 2002 and October 2006, with a declared value of about $10 million
PHILLIPINES
3 consignments between June 2005 and December 2005, with a declared value of over $250,000
MALAYSIA
48 consignments between September 2002 and September 2003, with a declared value of about $5 million
INDONESIA
21 consignments between October 2003 and September 2006, with a declared value of about $2 million

Chinese exports took a hit in 2002, when its honey was found tainted with the antibiotics, chloramphenicol. Border customs inspectors in some EU countries and Canada detected this antibiotic, a potential carcinogen; about 30 such consignments were detected in Europe. Other broad-spectrum antibiotics like streptomycin and tetracycline and pesticides like lindane were also found. The same year, the EU banned all honey imports from China.

This was followed by complaints from domestic honey growers of the US, including the American

to dodge US customs. This practice has come to be known as honey laundering.

What did the Indian government do?

As India began to come into focus for honey laundering, it took a step to ensure that exports were not affected and another ban was not imposed. The Directorate General of Foreign Trade of the ministry of commerce and industry issued a notification in February 2011. The notification imposed certain restrictions on honey exports to the EU and US. According to the notification,

honey exported shall be wholly obtained from Indian origin and that no blending will be permitted with honey originating from other countries.

At the time the notification was issued, the EU ban on Indian honey was still there. But what stands out is that Chinese honey is not banned in India but it cannot be re-exported to another country, especially the EU and the US. And in the absence of any rules in India on antibiotics in honey for domestic consumption, the Chinese honey that could be tainted with antibiotics and heavy metals is being blended and sold within India.[1]

those who eat these food animals.

The US sets the pace for antibiotic use in farm animals. Its meat industry has fed medically important antibiotics to chickens, pigs, and cattle to accelerate their weight gain and prevent disease in the stressful and unhygienic conditions that typify industrialised animal agriculture production facilities.[3] In 1951, the US Food and Drug Administration (FDA) approved the addition of penicillin and tetracycline to chicken feed as growth promoters, encouraging pharmaceutical companies to mass-produce antibiotics for animal agriculture.[4] By the 1970s, nearly 100 per cent of all birds commercially raised for meat in the US were being fed antibiotics.[5] By the late 1990s, poultry producers were using 5 million kg (11 million pounds) of antibiotics annually, more than a 300 per cent hike over the 1980s.[6]

The thousands of tonnes of antibiotics used in animal agriculture are typically not for treatment of sick and diseased animals. Rather, the drugs are used for non-therapeutic purposes. More than 90 per cent of US pig farms, for example, feed the animals antibiotics for such non-treatment reasons as weight gain.[7]

Though there is no data on the use of antibiotics in farm animals in India, there is evidence of livestock owners using oxytetracycline in cattle and poultry to promote growth.

7. What are health impacts of antibiotics in food?

Antibiotics used in food animals (which are sources of milk, meat, eggs and honey) can affect the public health because of their secretion in edible animal tissues in trace amounts usually called residues. For example, oxytetracycline[8] and chloramphenicol[9] residues have been found in honey. Some drugs have the potential to produce toxic reactions in consumers directly, while some others are able to produce allergic or hypersensitivity reactions.[10] For example, beta-lactam antibiotics (like ampicillin) can cause cutaneous eruptions, dermatitis, gastro-intestinal symptoms and anaphylaxis at very low doses. Such drugs include the penicillin and cephalosporin groups of antibiotics.

Indirect and long-term hazards include microbiological effects, carcinogenicity, reproductive effects and teratogenicity. Microbiological effects are one of the major health hazards in human beings. Antibiotic residues consumed along with edible tissues like milk, meat, eggs and honey can produce resistance in bacterial populations in the consumers.

These bacteria might then cause difficult-to-treat human infections. Certain drugs like nitrofurans and nitroimidiazoles can cause cancer in human population. Similarly, some drugs can produce reproductive and teratogenic effects at very low doses consumed for a prolonged period of time. The WHO, jointly with the Food and Agriculture Organization (FAO), has identified links between antibiotic use in animals and the emergence of mainly food-borne bacteria, which are resistant to important antibiotics used in treating infectious diseases in humans.

8. What is antibiotic resistance?

Antibiotic resistance is the ability of a microorganism to withstand the effects of an antibiotic. If an antibiotic is not effective, even in large doses, in treating an infection, then the microorganism that is responsible for the infection is declared as resistant to that antibiotic.[11]

The primary cause of antibiotic resistance is long-term over-exposure to antibiotics through their use as medicines in humans, as well as in animals, horticulture and for food preservation. The types of antibiotics used in animals are frequently the same as, or closely related to, those used in humans. Factors influencing the development of resistance include drug concentration, duration of exposure, organism type, antimicrobial type and host immune status.

9. How serious is the problem of antibiotic resistance?

Antibiotic resistance is a global public health concern today. The US Centers for Disease Control and Prevention (CDC) has described antibiotic resistance as "one of the world's most pressing health problems" because the "number of bacteria resistant to antibiotics has increased in the last decade

(and)... many bacterial infections are becoming resistant to the most commonly prescribed antibiotic treatments".[12] The WHO has also identified antibiotic resistance as "one of the three greatest threats to human health".[13]

Antibiotic resistance in bacteria evolves via natural selection through random mutation. When a bacterium is exposed to an antibiotic, it starts making changes in its DNA to withstand the effects of the medicine. Once it acquires a specific antibiotic-resistant gene, it quickly passes it on to its next generation. Nature has developed different systems for transfer of genes between bacteria (conjugation, transformation, transduction and transposition) and these mechanisms have proven effective in the promotion of resistant genes. If a bacterium carries several resistant genes, it is called multi-resistant or, informally, a superbug (see Box: *The superbug saga*).

10. What are the global evidences of antibiotic resistance?

- Evidence has emerged of an association between use of antibiotic agents in food animals and antibiotic resistance among bacteria isolated from humans. An outbreak of human nalidixic acid-resistant *Salmonella typhimurium* DT104 infection in Denmark was traced to a pig farm. Another outbreak of the same infection, reported in the United Kingdom, was traced to a dairy farm where fluoroquinolone had been used on the cattle a month before the outbreak.
- In the US, there was a marked increase in the proportion of domestically acquired Campylobacter infections that were fluoroquinolone-resistant, following the first approved use of fluoroquinolones in food animals in 1995.[14]
- The US FDA concluded that the use of antibiotics like ciprofloxacin in chicken production had compromised the treatment of nearly 10,000 Americans annually, meaning that thousands of people infected with Campylobacter who sought medical treatment were initially treated with an antibiotic to which the bacteria was resistant, forcing the doctors to switch to more powerful drugs.[15] A study involving thousands of patients with Campylobacter infections showed that resistant strains led to up to nearly 10 times more complications — including infections of the brain, the blood, and death.
- Antibiotic-resistant Salmonella has also led to serious human medical complications.[16] Food-borne Salmonella emerged in the US northeast in the late 1970s and has since spread throughout North America. One theory holds that multi drug-resistant Salmonella was disseminated worldwide in the 1980s via contaminated feed made out of farmed fish that had been fed routine antibiotics.[17]
- Alarmingly high rates of methicillin-resistant *Staphylococcus aureus* (MRSA) in farm animals and retail meat in Europe has led to increased scrutiny of agricultural use of antibiotics. The 2008 discovery of MRSA in North American pigs suggests the potential public health risk attributed to farm animal-associated MRSA may be a global phenomenon.[18]

11. What is the world doing to tackle this menace?

In the past, the WHO had recommended that antibiotics, which are also licensed in human medicine, should not be used any more as growth promoters in livestock. An EU resolution to this effe was introduced in 1999. On January 1 2006 the EU banned feeding of all antibiotics and related drugs t livestock for growth promotion purposes.[19]

Some EU member states (such as Denmark)

THE SUPERBUG SAGA

A drug-resistant bacterium dubbed the superbug triggered a debate in 2010. A gene – New Delhi metallo-beta lactamase (NDM 1) – transformed some bacteria found in the gut, like Escherichia coli and Klebsiella pneumoniae, into superbugs.

According to a study published in August 11 issue of science journal Lancet Infectious Disease, the NDM 1 was resistant to all antibiotics, except tigecycline and colistin. The study, based on a survey conducted in India, Pakistan and the UK, ended with an advisory to patients in the UK that they should not opt for surgeries in India.[1]

In June 2010 the superbug killed a Belgian patient who underwent treatment in Pakistan. The World Health Organization asked governments to focus on prevention and control through surveillance for anti-microbial resistance, rational use of antibiotics and preventing sale of antibiotics without prescriptions.

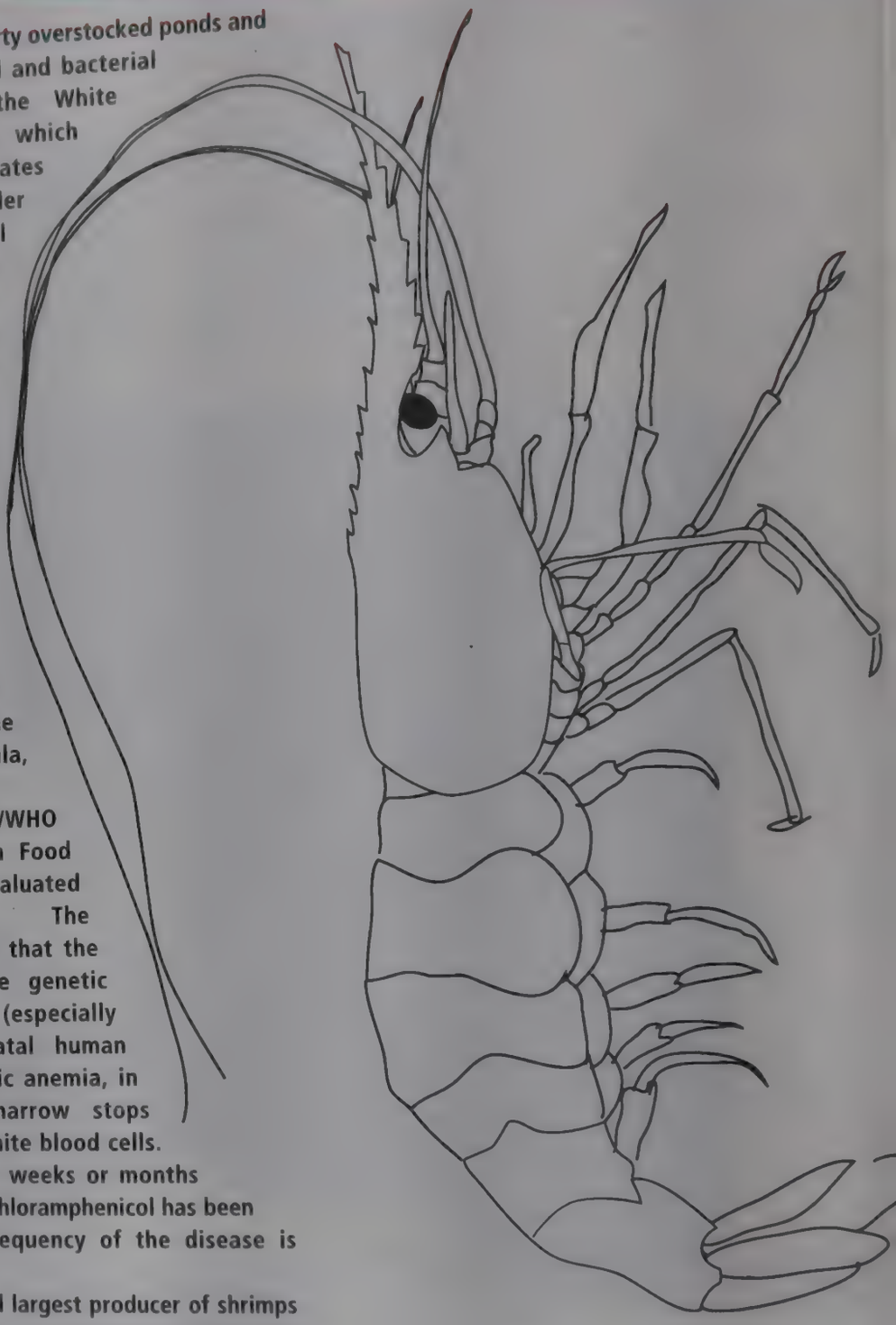
In its April 2011 edition, Lancet Infectious Disease published a paper which stated that the gene was found in tap water, rivulets and wate pools in Delhi. The gene makes the microbe tha carries it resistant to antibiotics.

have, with insignificant or no consequence either on disease rates in animals or on meat market prices, voluntarily suspended the use of all growth promoters irrespective of their human health importance.[20]

The US FDA, in November 2011, refused to ban the use of antibiotics in animals. It denied two citizen petitions that asked the agency to ban use of antibiotics given to animals in feed or water for purposes other than treatment of diseases and withdraw approval for most uses of penicillin and tetracyclines in animal feed. The FDA argued that withdrawing the antibiotics would be an expensive and a resource-intensive process.[21]

The Australian Pesticides and Veterinary Medicines Authority (APVMA) approves antibiotics for specific uses, such as to treat bacterial diseases or to prevent birds getting a disease when there is a high risk of this happening. The APVMA sets withholding periods for the approved uses. The Australian government tests for antibiotic residues as part of its National Residue Survey Program and no antibiotic residues have been detected in poultry products for years.[22]

ANTIBIOTICS IN SEAFOOD



Shrimps are farmed in dirty overstocked ponds and they get sick with viral and bacterial infections such as the White Spot Syndrome Virus, which causes high mortality rates throughout Asia. In order to keep them alive till they are sold, a range of antibiotics is used in shrimp farms. The most commonly used is chloramphenicol. Chloramphenicol is considered to be a drug of last resort for humans, usually administered only in life-threatening situations when less potent antibiotics are ineffective (for instance, in the treatment of salmonella, anthrax, and typhoid).

The Joint FAO/WHO Expert Committee on Food Additives has evaluated chloramphenicol. The committee concluded that the compound can cause genetic damage, cancer (especially leukemia) and a fatal human disease called aplastic anemia, in which the bone marrow stops producing red and white blood cells. The onset may occur weeks or months after treatment with chloramphenicol has been discontinued. The frequency of the disease is greatest in Asia.[1]

India is the second largest producer of shrimps in the world. In 2002, EU inspectors ordered the destruction of three large consignments of shrimp from India after chloramphenicol was detected. Chloramphenicol is banned in India for use in aquaculture.

In 2009, in the period from January to July, the EU rejected more than 50 shrimp consignments from India due to detection of an antibiotic residue, presence of bacteria, traces of heavy metals and unhygienic condition of the products. The total number of rejections in 2008 was around 30 . These rejections have hit exporters, especially since EU is their largest market.

The Food and Veterinary Office (FVO) mission of the European Commission, during its visit to India in 2009, observed that "there was no effective control on the distribution and use of veterinary medicinal products, particularly in aquaculture farms, dairy farms and apiaries, as evidenced by the high frequencies of non-compliant results in the national residue control plan and the RASFF (Rapid Allert System for Food and Feed) alerts for crustaceans".[2]

12. *How does India regulate antibiotics in food?*

In India, antibiotics are used widely in food animals as growth promoters and to prevent and treat infections. Non-therapeutic usage of antibiotics has been especially common in poultry production and fisheries and now, in beekeeping. However, under the Food Safety and Standards Act, 2006, currently there are no regulatory provisions regarding the use of antibiotics in livestock.

The Act does regulate the use of antibiotics in fisheries under its Regulation 4.4.2: Antibiotic and Other Pharmacologically Active Substances: "The amount of antibiotics for sea foods including shrimps, prawns or any other variety of fish and fishery products, shall not exceed the prescribed tolerance limit (mg/Kg [ppm]) as mentioned: Tetracycline (0.1), Oxytetracycline (0.1), Trimethoprim (0.05), Oxolinic acid (0.3)."

The Act gives a list of antibiotics and other pharmacologically active substances that are prohibited in any unit processing sea foods including shrimps, prawns or any other variety of fish and fishery products. They are Nitrofurans, Chloramphenicol, Neomycin, Nalidixic Acid, Sulphamethoxazole, Aristolochia spp. and preparations thereof, Chloroform, Chlorpromazine, Colchicine, Dapsone, Dimetridazole, Metronidazole, Ronidazole, Ipronidazole, other Nitroimidazoles, Clenbuterol, Diethylstilbistrol, Sulphonamide drugs, Fluoroquinolones and Glycopeptides (see Box: *Antibiotics in seafood*).

In March 2011, the Union ministry of health and family welfare formulated the National Policy for Containment of Antimicrobial Resistance. This policy has acknowledged the fact that antibiotics are used as growth promoters and to prevent and treat infections and also, that there are no regulatory provisions regarding the use of antibiotics in livestock. The policy points out that there is a need to regulate the use of antibiotics in poultry and other animals as well and put in place a requisite labelling requirement in food. The policy calls for a ban on the use of antibiotics in livestock for non-therapeutic uses.

At the moment, the policy is on hold pending discussions.

13. *What has been the impact of the CSE study on the way India regulates honey?*

Following the CSE study, the Rajya Sabha – in the parliament's winter session in 2010 – raised the issue of antibiotics in honey. The then minister of state for agriculture, consumer affairs, food and public distribution, K V Thomas, in a written reply to the parliament acknowledged the fact that the European Union had banned the export of honey from India on account of positive detection of heavy metals and other contaminants.

The Food Safety and Standards Authority of India (FSSAI) was quick to react to the CSE study, but all it did was issue an advisory in September 2010. The advisory stated that no antibiotics and pesticide residues would be allowed in honey. It added that with regard to "antibiotics in honey, the safety standards in India are similar to the rules in the European Union, Codex Alimentarius and USA where they are completely prohibited". The advisory also specified maximum limits for heavy metals in honey.

Around December 2010, the government directed the Bureau of Indian Standards (BIS) to analyse the CSE study. The 32-member technical committee of the BIS met on January 5, 2011 to revise the Indian Standard Specifications for extracted honey. The committee, after due deliberations, agreed that clause 5.4 of IS 4941:1994 'Extracted Honey – Specification (second revision)' needs to be revisited and that no antibiotics should be tolerated in honey. The clause pertains to the absence of additives in honey; these additives include pesticides, antibiotics and heavy metals. The BIS, however, has not yet completed its work in this matter.

In August 2011, the FSSAI's scientific panel for pesticides and antibiotic residues (in its third

WHAT, THEN, IS THE WAY AHEAD?

- **The government should promote good practices through universities, horticulture board and regulatory agencies.**
- **The government should put standards regulating the use of antibiotics in place.**
- **The government should monitor the presence of antibiotics in food items the same way that pesticide residues are monitored through the All India Network Project on Pesticides Residue.**
- **The government should put in place the regulations to control the sale of antibiotics and also the punitive action that needs to be taken in case the regulations are violated.**
- **The strict enforcement of law and complete transparency of the enactment of the regulations should be ensured.**

FSSAI RECOMMENDATIONS FOR TESTING OF PESTICIDES IN HONEY

Antibiotics	LOQ (ppb)	Instrument used
Chloramphenicol	0.3	LC-MS/MS
Nitrofurans	0.5	LC-MS/MS
Sulponamides	5.0	LC-MS/MS
Streptomycin	5.0	LC-MS/MS
Tetracycline	5.0	LC-MS/MS
– Oxytetracycline	5.0	LC-MS/MS
– Chlortetracycline	5.0	LC-MS/MS
Ampicillin	5.0	LC-MS/MS
Enrofloxacin	5.0	LC-MS/MS
Ciprofloxacin	5.0	LC-MS/MS
Erythromycin	5.0	LC-MS/MS
Tylsoin	5.0	LC-MS/MS

Source: The Food Safety and Standards Authority of India (FSSAI)

meeting) began discussing antibiotics in honey. Taking cognizance of the CSE report, the panel noted that there was enough evidence of antibiotics in honey and that there was a need for a well designed study, uniform risk assessment methods and a study on consumption patterns of honey by the vulnerable age groups. It was also mentioned that while use of the tetracycline class of antibiotics had been allowed under some conditions, the rest of antibiotics that were being used were to be considered merely contaminants. The panel also cautioned that fixing MRLs of antibiotics in honey would actually give the manufacturers a license to openly use antibiotics in honey production.

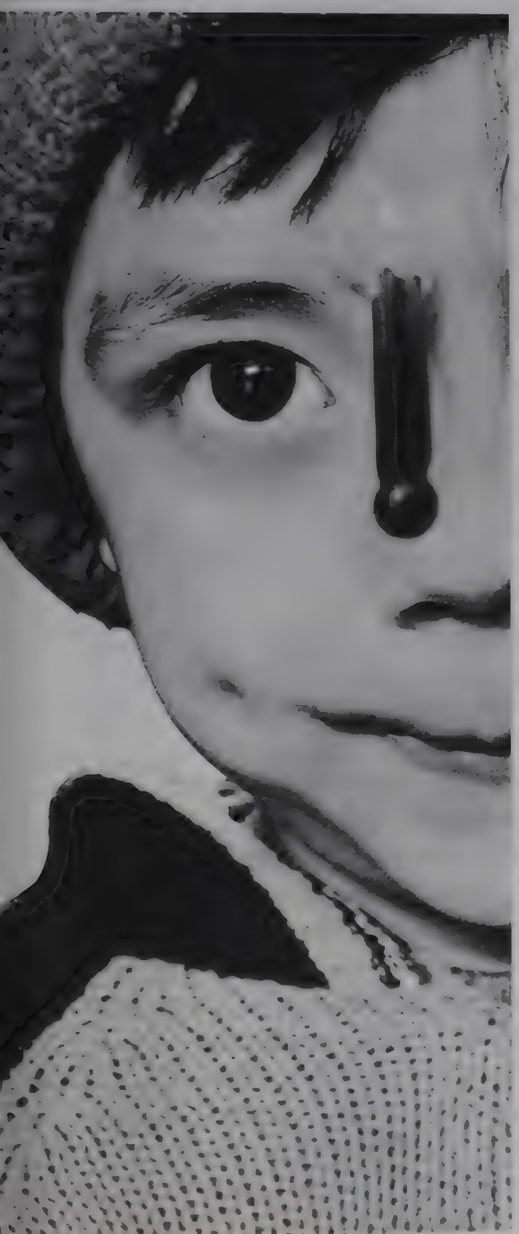
In October 2011, the FSSAI scientific panel decided to follow the EU norms for antibiotic residues; for those antibiotics which were not included under the EU norms, such as ciprofloxacin, enrofloxacin and ampicillin, their LOQs (Limits of Quantification) were to be identified. It was decided to also set the limits for the other antibiotics that were being used in India in beekeeping. It was decided in principle that antibiotics in honey should not be used at any stage.

After a few more meetings in December 2011, the FSSAI scientific panel recommended a list of antibiotics with their LOQs determined by the LC/MS technique. This list included the antibiotics that CSE had tested for (see Table: *FSSAI recommendations for testing of pesticides in honey*). The FSSAI, however, is yet to take a decision on the final standards.●

TOXINS AT HOME

3

Modern households are full of harmful chemicals. From the paints on walls and furnitures to detergents, toys, water bottles, room fresheners, cosmetics, packaging etc, toxic chemicals are lurking everywhere. They can cause cancer, hypertension, asthma, skin allergy and many more aliments. They can make a child's IQ lower or make children hyperactive and concentration-deficient.



But we do not know about them because labels on household products in India do not give their chemical compositions or any warning of what toxins such products may contain. They are not bound to, as most everyday household products have only voluntary standards.

In India, almost all regulations for chemicals are process regulations, not product regulations: the factory must comply with the discharge standards, but the chemical composition of the product that leaves the factory remains unregulated. In fact, there is no chemical registry – a register of all chemicals produced, imported, exported and used in the country. Chemicals that are banned in many countries because of health and environmental toxicity are freely produced and used in India because there is no one to regulate and control.

It is high time that product standards are mandatorily set for products and harmful chemicals removed from our daily lives.

3.1 LEAD IN PAINTS

PHOTOGRAPHS: MEETA AHLAWAT / CSE

1. What are paints made up of? Why is lead used in paints?

Paint is composed of two basic ingredients: pigment and a binder. A solvent (thinner) is used to adjust the application and properties of the paint. The solvent traditionally makes up about 60 per cent of the total formulation but does not become part of the paint film. Binders account for 30 per cent, pigments make up 7 to 8 per cent, and additives 2 to 3 per cent. Opaque pigments give the paint its hiding or covering capacity and contribute other properties.

Lead is one of the 300-odd ingredients used to make paints. It is silvery in its purest form, but turns blue-grey when exposed to air. Lead is primarily used in paints as a pigment to impart color. It is also added to make paints more durable and corrosion resistant, and for speed drying. Lead chromate (also called chrome yellow) and lead carbonate (also called white lead) are widely used as pigments. Lead chromate is often used to make yellow, orange, red and green paints. Lead carbonate is a superior paint pigment used for its opaque quality. Other pigments like red lead and blue lead impart corrosion protection as well as color to metal surface.

2. Why is lead harmful?

Lead is one of the most common health hazards young children face today. Among the medical fraternity, it is referred to as the 'silent epidemic'. The human body is not designed to process lead. Sustained and large exposures can cause serious damage to health.

One of the first cases of lead poisoning was documented way back in 1887 when the US medical authorities found lead toxicity symptoms in children. In 1904, a physician, James Lockhart Gibson, linked child lead poisoning to paints for the first time in an article in the *Australian Medical Gazette*.

Segments of the population at the highest risk of health effects from lead exposure are pre-school age children, pregnant women and their fetuses. Children are especially susceptible to lead poisoning (see Box: *Lead in their blood*). Until five years of age, they can absorb up to 50 per cent of the lead they ingest. Once inside their body, it begins damaging their central nervous system and brain. Adults may find it difficult to concentrate or remember things and feel pain in muscles and joints (see Figure: *The infiltrator*).

LEAD IN THEIR BLOOD

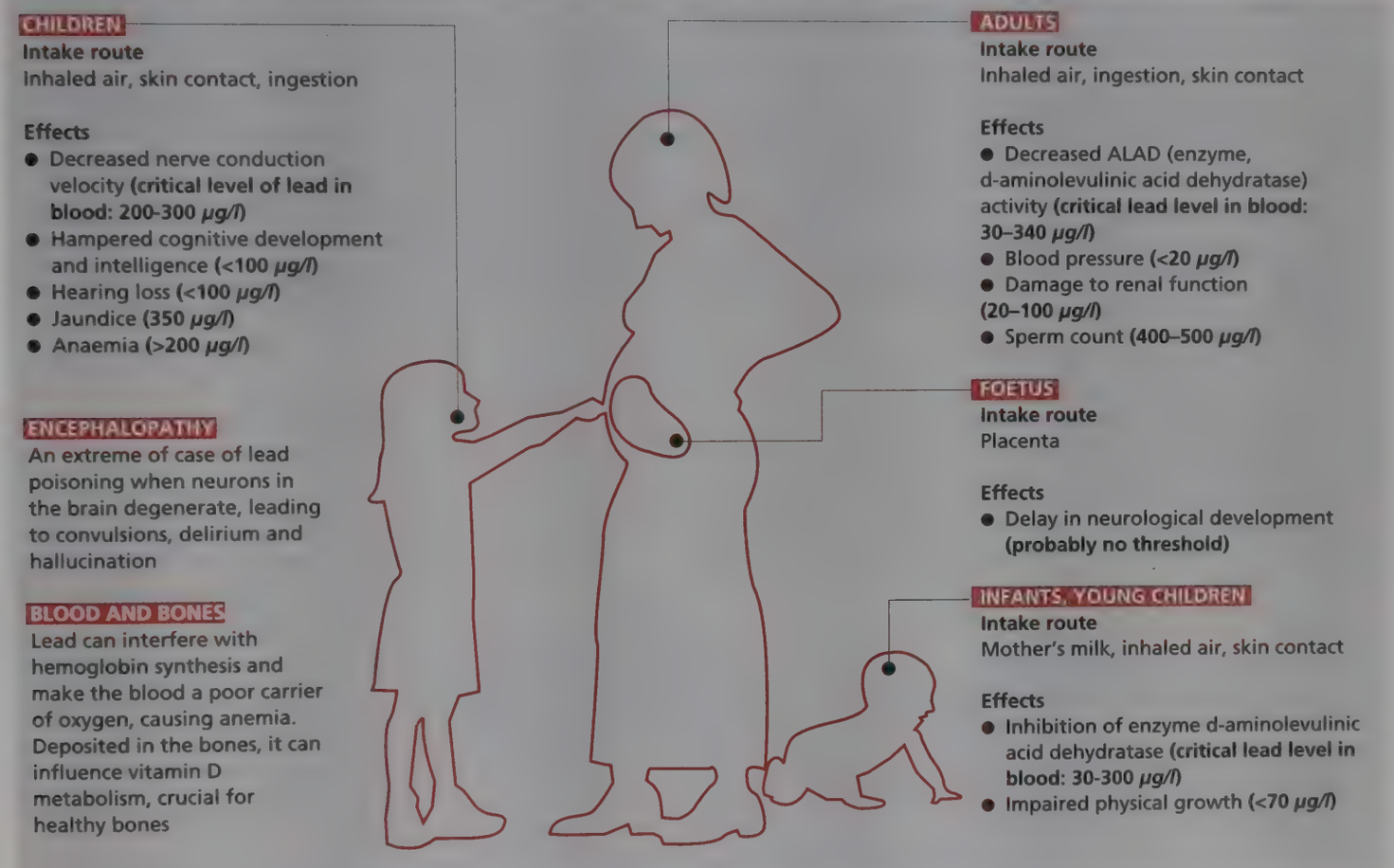
Studies have shown over 60 per cent children in India may have more than 10 microgramme per deciliter (mg/dl) lead in their blood. In 1999, the George Foundation, a non-profit in Bengaluru, took blood samples of schoolchildren in some major Indian cities. It found over 52 per cent had more than 10 mg/dl lead in their blood.

In 2004 researchers from Kasturba Medical College in Mangalore found evidence of lead ingestion through paints. They studied lead levels in the blood of children and in their surroundings. The results were published in the Indian Journal of Pediatrics in 2004. Of the 104 children studied, 11 had over 40 mg/dl of lead concentration. One, who had a blood lead level of 72.7 mg/dl, regularly played in a public park, on a swing coated with a lead-based paint. The paint was pealing off.

The researchers asked the municipal officials to remove the paint and use another one without lead. A follow-up examination of the child three weeks later showed that the lead level in his blood had gone down to 45.9 mg/dl.[1]

THE INFILTRATOR

Lead harms us at every stage of development, but childhood is the most vulnerable


CHILDREN
Intake route
Inhaled air, skin contact, ingestion
Effects
● Decreased nerve conduction velocity (critical level of lead in blood: 200-300 µg/l)
● Hampered cognitive development and intelligence (<100 µg/l)
● Hearing loss (<100 µg/l)
● Jaundice (350 µg/l)
● Anaemia (>200 µg/l)
ENCEPHALOPATHY
An extreme of case of lead poisoning when neurons in the brain degenerate, leading to convulsions, delirium and hallucination
BLOOD AND BONES
Lead can interfere with hemoglobin synthesis and make the blood a poor carrier of oxygen, causing anemia. Deposited in the bones, it can influence vitamin D metabolism, crucial for healthy bones
ADULTS
Intake route
Inhaled air, ingestion, skin contact
Effects
● Decreased ALAD (enzyme, d-aminolevulinic acid dehydratase) activity (critical lead level in blood: 30–340 µg/l)
● Blood pressure (<20 µg/l)
● Damage to renal function (20–100 µg/l)
● Sperm count (400–500 µg/l)
FOETUS
Intake route
Placenta
Effects
● Delay in neurological development (probably no threshold)
INFANTS, YOUNG CHILDREN
Intake route
Mother's milk, inhaled air, skin contact
Effects
● Inhibition of enzyme d-aminolevulinic acid dehydratase (critical lead level in blood: 30-300 µg/l)
● Impaired physical growth (<70 µg/l)

Source: *Down To Earth*, August 15-31, 2009

3. How is lead ingested by the human body?

It is easy to expose oneself to lead; you may pick it up while admiring the velvety touch of paint on walls or when walking past a car belching exhaust if it is using leaded fuel. Some dyes, batteries, smelting units and soldering on water pipes contain lead. The most common way of lead poisoning is inhaling lead dust. Everyday activities like opening or closing windows glossed with lead-based paints or walking on leaded paint chips can raise lead dust. The dust can float in the air we inhale or settle on the floor and food.

Occupational hazards (for example, those experienced by painters and abatement workers who use unsafe paint removal practices) are the leading causes of elevated blood lead levels in adults. Ingestion of lead-contaminated surface dust is the most common pathway of lead poisoning in children.

4. What were the key findings of the CSE study on lead in paints?

CSE's Pollution Monitoring Lab tested Indian paints to find out how much lead they contained. Two sets of tests were carried out; the first tests took place from May to June 2008. A repeat test was carried out

WHAT THE CSE STUDY FOUND

Phase I: May-June 2008

- CSE procured 25 samples of popular enamel paints from Delhi markets and analysed them for lead content.
- The brands tested were Apcolite (Asian Paints), Nerolac (Kansai Nerolac Paints), Luxol (Berger Paints India), Superlac (Shalimar Paints) and Dulux (ICI India). The colours were yellow, orange, green, black and white.
- 72 per cent of these samples contained lead much higher than the 1,000 ppm specified by BIS.
- The lead concentration varied from zero to 184,733 ppm, the highest amount being present in the deep orange paint of Superlac brand.
- All the samples of Dulux brand (ICI) had lead much below the specified limit of 1,000 ppm. White shades of Asian Paints and Nerolac also conformed to the standards.
- In order of decreasing lead concentration paint colours can be arranged as: Yellow > Orange > Green > Black > White

Phase II: May-June 2009

- Eight more samples of four brands were procured and analysed. The results showed that deep orange and black paints of Apcolite brand (Asian Paints) contained 29.24 and 28.71 ppm lead, compared to 59,149 ppm and 17,720 ppm respectively found earlier. This indicated the company was trying to phase out lead.
- In case of golden yellow and tractor orange of Nerolac brand the lead concentration was 10.02 ppm and 1,991.9 ppm respectively as compared to 131,254 and 119,086 ppm found earlier.
- Two manufacturers, Berger and Shalimar, exceeded the lead content found in their paints in the earlier study.
- The golden yellow and bus green of Luxol brand (Berger Paints) showed a lead concentration of 310,721 ppm and 68,105 ppm. In case of golden yellow and black colours of Superlac (Shalimar Paints), the lead content was 287,062 ppm and 5,983.8 ppm.

in June 2009. After the first round of tests, CSE sent a questionnaire to the paint industry telling them about the findings of the study and asking them what they proposed to do. The companies got back after a few months saying that they had gone lead-free. So CSE tested for a second time in 2009 (see Table on p62-63: *Paint's toxicity card*).

Twenty five samples (five colors) of enamel paint made by the major players – Asian Paints (India), Goodlass Nerolac Paints (now known as Kansai Nerolac), Berger Paints, Shalimar Paints, and ICI (India) – were randomly purchased from markets in Delhi between November 2007 and May 2008 (see Box: *What the CSE study found*).

5. What are some of the regulations in place around the world?

Historically, many countries have investigated lead poisoning and recommended substitution with non-lead pigments.

Countries that have banned or restricted the use of white lead for paint include France, Belgium and Austria in 1909; Tunisia and Greece in 1922; Czechoslovakia in 1924; Great Britain and Sweden in 1926; Poland in 1927; Spain and Yugoslavia in 1931; and Cuba in 1934. In 1922 the Third International Labour Conference of the League of Nations recommended the banning of white lead for interior use.

A look at the regulations in various parts of the world:

USA: Lead-based paint is defined by statute as paint with lead levels equal or exceeding 0.5 per cent (5,000 ppm) or 1.0 mg/cm^2, according to the US Environment Protection Agency (EPA) Standards (40 CFR Part 745, 2001). For paint and similar surface coatings, and certain consumer products, 16 CFR § 1303.1 specifies that the maximum allowable total lead content is 0.06 per cent based on the total weight of the non-volatile portion of the paint (which is equivalent to 600 ppm).

The lead limit in paint and surface coatings applies to (i) paint and other similar surface coatings; (ii) toys and other articles intended for use by children; and (iii) certain furniture articles that are not otherwise exempt under our regulations. As of August 14, 2009, the maximum allowable total lead content of such items fell to 0.009 per cent (which is equivalent to 90 ppm).

Canada: Under new regulations published in the *Canada Gazette Part II* on May 4, 2005, the lead content limit has been reduced from 5,000 mg/kg (0.5 per cent by weight) to 600 mg/kg (0.06 per cent by weight) for surface coating materials used in or

around the home or other premises where children or pregnant women may become exposed, or on furniture, toys and other articles for children, as well as on pencils and artist's brushes.

European Union: In 1988, an 8th amendment to the Marketing and Use Directive 76/769/EEC proposed a complete ban on the use of lead carbonate and sulphate pigments (white lead) in paints. However, in September 1989, the European Parliament permitted derogation in the Marketing and Use Directive (89/677/EEC) allowing the use of leaded paints for works of art and historic buildings. The Directive stated that lead carbonates and sulphate "may not be used as substances or constituents of preparations intended for use as paints, except for the restoration and maintenance of works of art and historic buildings and their interiors, where member states wish to authorize this on their territory, in accordance with the provisions of ILO Convention 13 on the use of white lead in paint".

The 1999/45/EC of the European parliament and of the council of May 31, 1999 concerning the labeling of dangerous preparations states that "Labels of paints and varnishes containing lead in quantities exceeding 0.15% (expressed as weight of metal) of the total weight of the preparation as determined in accordance with ISO standard 6503/1984 must show the following particulars 'Contains lead. Should not be used on surfaces liable to be chewed or sucked by children'."

Australia: The recommended amount of lead in domestic paints has declined from 50 per cent before 1965, to 1 per cent in 1965. In 1992, it was reduced to 0.25 per cent and in 1997, to 0.1 per cent.

Singapore: Singapore has a standard for lead compounds in paints in the list of hazardous substances controlled under the Environment Protection and Management Act (EPMA), which states that the lead content should not be more than 0.06 per cent (600 ppm) by weight of the paint.[1]

Sri Lanka: This is the latest country to introduce standards for lead in paints. The Consumer Affairs Authority published a Gazette order on September 30, 2011 regulating permissible maximum lead content on paints and accessories. It shall come into force from January 1, 2013.

The Gazette states that "no Manufacturer, Importer, Packer, Distributor or Trader shall manufacture, import and use or distribute, pack, store or sell or display for sale, expose for sale or offer for sale, wholesale or retail any paints unless such paints shall conform to the corresponding Total Lead Content given hereunder as specified by the Sri Lanka Standard Institution for such paints".

Sri Lankan authorities have set the permissible limits for different categories. For instance, the permissible limit in paints for toys and accessories for children is 90 mg/kg (90 ppm); enamel paints and floor paints are set at 600 mg/kg (600 ppm), while

PAINTS TOXICITY CARD

Concentration of lead in enamel paints sold in Delhi. CSE analysed them in two phases in 2008 and 2009

Brand name	Colour	Company	2008			
			Lead (ppm)	Lead (%)	No of times of the BIS limit (IS 5489:2004) (1,000 ppm)	No of times US paints lim (600 ppm)
Apcolite	Golden yellow	Asian Paint	91,327	9.13	91	152
Nerolac	Golden yellow	Nerolac	131,254	13.13	131	219
Luxol	Golden yellow	Berger	162,559	16.26	163	271
Dulux	Golden yellow	ICI	0.00	0.000	within limit	within limit
Superlac	Golden yellow	Shalimar	151,212	15.12	151	252
Apcolite	Deep orange	Asian Paint	59,149	5.91	59	99
Nerolac	Tractor orange	Nerolac	119,086	11.91	119	198
Luxol	Deep orange	Berger	154,306	15.43	154	257
Dulux	Deep orange	ICI	0.00	0.000	within limit	within limit
Superlac	Deep orange	Shalimar	184,733	18.47	185	308
Apcolite	Bus green	Asian Paint	22,182	2.22	22	37
Nerolac	Bus green	Nerolac	23,591	2.36	24	39
Luxol	Bus green	Berger	25,008	2.50	25	42
Dulux	Bus green	ICI	21.76	0.002	within limit	within limit
Superlac	Bus green	Shalimar	35,330	3.53	35	59
Apcolite	Black	Asian Paint	17,720	1.77	18	30
Nerolac	Black	Nerolac	11,141	1.11	11	19
Luxol	Black	Berger	17,488	1.75	17	29
Dulux	Black	ICI	36.25	0.004	within limit	within limit
Superlac	Black	Shalimar	14,863	1.49	15	25
Apcolite	BLZ white	Asian Paint	82.02	0.008	within limit	within limit
Nerolac	Brill white	Nerolac	11.65	0.001	within limit	within limit
Luxol	Snow white	Berger	2,133.2	0.21	2	4
Dulux	Blazing white	ICI	9.44	0.001	within limit	within limit
Superlac	Dazzling white	Shalimar	3,461.1	0.35	3	6

Source: *Down To Earth*, August 15-31, 2009

emulsion paints for exterior and interior use is set at 90 mg/kg (90 ppm).[2]

6. How does India regulate its paints industry?

The Bureau of Indian Standards (BIS) has set voluntary standards for presence of lead in paint and is governed by IS 15489:2004-Paints, Emulsion Specification. The Specific Requirements of Standard IS 15489 : 2004 in subsection 6.12.2.2 states that , "The product shall not contain more than 0.1 percent by mass (as metal), of any toxic metals such as lead, cadmium, chromium (VI) and their compounds when tested by the relevant Atomic Absorption Spectrophotometric methods".

This standard also includes a scheme for labeling environment friendly products known as Eco Mark introduced by the ministry of environment and forests (MoEF) and administered by the BIS under the BIS Act, 1986 as per Resolution No 71 dated February 20, 1991, and published in the Gazette of Government of India.

As the requirement for lead is to be below 0.1 per cent (1,000 ppm), this comes under the optional scheme of Eco Mark: thus, a paint product labeled ISI (thereby confirming to the BIS voluntary standards) may not contain lead below 1,000 ppm unless it also has Eco Mark.

To qualify for the label, paints have to have less

2009			
ead ppm)	Lead (%)	No of times of the BIS limit (IS 5489:2004) (1,000 ppm)	No of times of the US paints limit (600 ppm)
.02	0.001	within limit	within limit
0,721	31.07	311	518
37,062	28.71	287	478
9.24	0.003	within limit	within limit
991.9	0.20	2	3
8,105	6.81	68	114
8.71	0.003	within limit	within limit
,983.8	0.60	6	10

than 1,000 ppm lead and no petroleum derivate comprising volatile organic compounds. Till 2009 end, no paint company had applied for eco-marking their paints, according to the Central Pollution Control Board, which monitors the Eco Mark.

As the BIS standard is voluntary in nature (unlike certification), paint manufacturers need not necessarily meet BIS specifications and the Indian paint industry is virtually unregulated for lead content.

7. What are the substitutes that can replace lead in paints?

Over the years, paint manufacturers replaced white lead with a less toxic substitute, titanium white (based on the pigment titanium dioxide) which was first used in paints in the 19th century. It is considered safe enough to be used as a food coloring and in toothpaste, and is a common ingredient in sunscreen. The titanium white used in most paints today is often coated with silicon or aluminum oxides for better durability.

Organic pigments are also used as alternatives. Then there is zirconium that can be mixed in paints for quicker drying and gloss. The barium-zinc-sulfur combination replaces white lead.

8. How big is the Indian paints industry?

The organised sector, represented by six major players, controls 70-75 per cent of the paint market in India. The unorganised sector comprises 2,000-odd small formulators. Asian Paints is the leader, with over 40 per cent share of the organised market. Kansai Nerolac with 20 per cent, Berger Paints with 17 per cent and ICI with 12 per cent are the next biggest players.

The decorative segment makes up the bulk of India's paint industry – roughly 75 per cent. But the industrial paint segment growth is likely to be spurred by a boom in the automotive and construction industries.

The per capita consumption of paints in India is still low at 1.25 kg against 51.7 kg in Qatar, 38 kg in Singapore or 25.8 kg in the US. Even China has a per capita consumption of 2.5 kg and Sri Lanka, 3.5 kg.

The industry in India produces approximately 940 million liter, valued at about US $2 billion. The organised sector contributes 54 per cent of the total volume and 65 per cent of the value.

Over the last 10 years, the industry has grown at a compounded annual growth rate (CAGR) of 12-13 per cent.[3]

9. Can the Indian paint industry invest in lead-free products? What are the technological and cost imperatives of making paint lead-free?

According to the Indian Paint Research Centre in Kolkata, all companies in India have the capacity to manufacture paint without lead. There is a price difference between lead and lead-free paint of the same shade but it is not substantial.

ICI's Dulux is the only brand that does not use lead in its formulations. Asian Paints and Nerolac claim to have phased out lead in their paints since April 2008. Though the CSE study in 2009 showed near-zero lead in Asian Paints samples, Nerolac Paints still contained a little lead. Asian Paints has removed lead from the decorative segment of paints primarily sold for residential purposes; only a few paints in the industrial sector still have lead.

However, since India has many small scale paint manufacturers that produce cheap lead-based paints there is a price pressure on other large manufacturers not to move to lead-free paints. For instance, ICI claimed to be losing its market because it was the first company to switch over to lead-free paints.

It is, therefore, important that mandatory standards are set providing level playing field to all players.

10. What does the Indian government need to do?

The government needs to set mandatory and stringent rules to ensure that paints become lead free. Though in the course of the CSE study, a couple of paint manufacturers did claim to bring down the lead content in their products, but that was only for the decorative paint segment.

There is a need to make labeling laws stringent ensuring that the paint manufacturers carry a warning about lead. Of the samples that CSE had tested only one brand, Dulux, had a label stating "No added lead, mercury, chromium compounds around a mark of green tree". Dulux was the only brand that had lead levels below the voluntary 1,000 ppm mark.

11. What is the regulation that the Indian government is working upon?

The Bureau of Indian Standards has prepared a draft notification on lead standards for all the categories of paints used for household and decorative purposes. For most of the paints the maximum lead limit has been proposed at 90 ppm, which is the current global best practice. A few of the industrial paints are to have 300 ppm lead limit for technical reasons. The final notifications are yet to come.

On the other hand, the Department of Industrial Policy and Promotion (DIPP) of the ministry of commerce and industry constituted a sub-committee for regulating lead in paints. CSE is a member of this sub-committee, which has had just a couple of metings till date, without any outcomes. ●

3.2 PHTHALATES IN TOYS

They lurk inside plastics, and from there migrate to air, food, human body and even unborn babies. Phthalates or phthalate esters are organic chemicals commonly used as plasticizers to make plastic supple. They are responsible for plastic products being cheap, easy to clean and toxic.

CSE conducted a study in 2009 on toys and its phthalates content and found that all samples contained one or more phthalates – di-n-butyl phthalate (DBP), benzyl butyl phthalate (BBP), di (2-ethylhexyl) phthalate (DEHP) and diisononyl phthalate (DINP) – all harmful in varying concentrations.

These toxic chemicals are not regulated or monitored by the government, putting children at risk. Scientific evidence has shown that exposure to phthalates can cause a variety of health problems ranging from asthma to pre-term birth. It is for this reason that the US and European Union regulate the use of these substances in toys. However, India and China, the largest toy producers, did not regulate phthalates.

The CSE study and pressure from various groups set the ball rolling and the BIS has now made voluntary rules for phthalates in toys. These rules, that are yet to be notified, need to be made mandatory.

1. *What are phthalates?*

Phthalates or phthalates esters are esters of phthalic acid mainly used as plasticisers (substances added to plastics to increase their flexibility) in poly vinyl chloride (PVC). PVC is used widely in toys and other children's products such as chewy teethers, soft figures etc (see Box: *Pthalate products*). While there are nearly 20 kinds of phthalates, some of them can have severe health impacts (see questions and their answers in the following pages).

Phthalates migrate into the air, food and people, including babies in their mother's wombs. They have been found in indoor air and dust, and in human urine, blood, and breast milk. Phthalates can be released from soft PVC by surface contact, especially where mechanical pressure is applied (during chewing of a PVC teether, for instance). Loss of phthalates by volatilisation over time from soft PVC has long been recognised.

Phthalates are not just present in toys. Release of phthalates during manufacture, use and disposal

PHTHALATE PRODUCTS

Phthalates are used in a large variety of products, from enteric coatings of pharmaceutical pills and nutritional supplements to viscosity control agents, gelling agents, stabilisers, dispersants, lubricants, binders, emulsifying agents etc.

End-applications include adhesives and glues, electronics, agricultural adjuvants, building material, personal-care products, medical devices, detergents and surfactants, packaging, children's toys, modeling clay, waxes, paints, printing inks and coatings, pharmaceuticals, food products, and textiles. The plastic rubber ducks floating in the bathtub are so squishy because of the phthalates with which they have been saturated.

Phthalates are also frequently used in soft plastic fishing lures, caulk, paint pigments and nail polish. They are used in a variety of household applications (shower curtains, adhesives and perfumes) modern pop culture electronics and medical applications such as catheters.

The distinctive new smell of the car comes from all the phthalates that have been added to the plastic dashboard. The reason that dashboards become more brittle as the car gets older is because the phthalates are slowly migrating out and into the interior of the car, and into your skin when you are in the car.[1]

The most widely used phthalates are the di-2-ethyl hexyl phthalate (DEHP), the di-isodecyl phthalate (DIDP), and the di-isononyl phthalate (DINP). DEHP is the dominant plasticiser used in PVC due to its low cost. Benzyl butyl phthalate (BBP) is used in the manufacture of foamed PVC, which is used as a flooring material.

of PVC products, in addition to their use as additives in inks, lubricants, perfumes and other open-ended applications, has led to their ubiquitous distribution and abundance in the global environment.

COMMON PHTHALATES AND THEIR STRUCTURAL FORMULAE

Name	Acronym	Structural formula
Dimethyl phthalate	DMP	$C_6H_4(COOCH_3)_2$
Diethyl phthalate	DEP	$C_6H_4(COOC_2H_5)_2$
Diallyl phthalate	DAP	$C_6H_4(COOCH_2CH{=}CH_2)_2$
Di-n-propyl phthalate	DPP	$C_6H_4[COO(CH_2)_2CH_3]_2$
Di-n-butyl phthalate	DBP	$C_6H_4[COO(CH_2)_3CH_3]_2$
Diisobutyl phthalate	DIBP	$C_6H_4[COOCH_2CH(CH_3)_2]_2$
Di-n-pentyl phthalate	DNPP	$C_6H_4[COO(CH_2)_4CH_3]_2$
Dicyclohexyl phthalate	DCP	$C_6H_4[COOC_6H_{11}]_2$
Butyl benzyl phthalate	BBP	$CH_3(CH_2)_3OOCC_6H_4COOCH_2C_6H_5$
Di-n-hexyl phthalate	DNHP	$C_6H_4[COO(CH_2)_5CH_3]_2$
Diisohexyl phthalate	DIHxP	$C_6H_4[COO(CH_2)_3CH(CH_3)_2]_2$
Diisoheptyl phthalate	DIHpP	$C_6H_4[COO(CH_2)_4CH(CH_3)_2]_2$
Di(2-ethylhexyl) phthalate	DEHP, DOP	$C_6H_4[COOCH_2CH(C_2H_5)(CH_2)_3CH_3]_2$
Di(n-octyl) phthalate	DNOP	$C_6H_4[COO(CH_2)_7CH_3]_2$
Diisooctyl phthalate	DIOP	$C_6H_4[COO(CH_2)_5CH(CH_3)_2]_2$
Diisononyl phthalate	DINP	$C_6H_4[COO(CH_2)_6CH(CH_3)_2]_2$
Diisodecyl phthalate	DIDP	$C_6H_4[COO(CH_2)_7CH(CH_3)_2]_2$
Ditridecyl phthalate	DTDP	$C_6H_4[COO(CH_2)_{12}CH_3]_2$
Diisotridecyl phthalate	DIUP	$C_6H_4[COO(CH_2)_{10}CH(CH_3)_2]_2$

Source: *Down To Earth*, January 15-31, 2010

2. *What did CSE find in its study on phthalates in toys?*

In 2008, the Pollution Monitoring Laboratory of CSE tested 24 toy samples of major brands – purchased randomly from markets in Delhi – for the presence of phthalates. Fifteen were soft toys and nine were hard toys made in four countries.

Tests showed all samples contained one or more phthalates – DBP, BBP, DEHP and DINP, all harmful – in varying concentrations (see Figure: *The results*). The lab also found that:

- Eleven samples (46 per cent) had phthalates exceeding the EU limit of 0.1 per cent by mass of plasticised material (The threshold limit cannot be set lower than 0.1 per cent as phthalates can be found below this level as contaminants in the manufacturing process even if not used as plasticisers).
- DINP was detected in nearly 42 per cent of the samples; in 29 per cent, it exceeded the EU limit. The highest concentration of DINP, which is restricted in the US and EU in toys that can be put in the mouth, was found in the squeaky toys made by the Indian company Funskool India. At 16 per cent concentration, it was 162 times the EU limit.
- DEHP was detected in 96 per cent of the toys but in concentrations below the EU limit, except in a teether and two toys. The baby teether ostensibly made of non-toxic, food-grade silicone rubber had DEHP at a concentration three times the EU limit. A company in Taiwan had made it.
- DBP was found in soft and hard biters at levels two times the EU limit.

- The majority of the toys which contained high levels of phthalates were made in China. Six squeeze toys from China contained phthalates two to 80 times above the EU limit. Four of these were made by Lovely Collection, which did not even bother to mention the address of the manufacturer and the date of manufacture on the package.

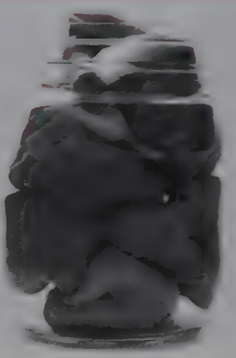
China, which has cornered 70 per cent of the global toy market, does not regulate the use of phthalates in toys. International standards dealing with toy safety ignore them. While the EU took the lead in imposing limits for phthalates in toys, the US has only recently passed a law.

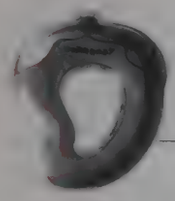
3. *What are the global evidences of health impacts due to phthalates?*

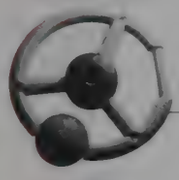
Phthalates can damage the male reproductive system, impair the lungs and affect the duration of pregnancy. Children under three years are more likely to be exposed to phthalates because they tend to chew and suck on plastic toys. Since their

THE RESULTS

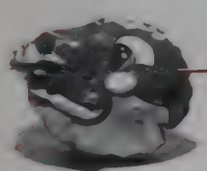
About half of the samples tested exceeded the internationally accepted safe limit for phthalates. Shockingly, toys meant for the most vulnerable, children less than three years, contained the highest amount of this toxin







Funskool



Puff 'N' Play

PiyoPiyo

Presence of phthalates

Category of toys	Number of samples	Percentage exceeding 0.1% individual phthalate limit
Toys put in mouth	7	29 (2)
Soft squeeze toys	9	67 (6)
Hard rattle toys	3	0 (0)
Others (dolls, ponies, etc)	5	60 (3)

Profile of toys with phthalates, based on country of origin

Country of manufacture	Number of samples	Percentage exceeding 0.1% individual phthalate limit
China	14	57 (8)
India	7	14 (1)
Taiwan	2	100 (2)
Thailand	1	0 (0)

Presence of phthalates among hard and soft toys

Category of toys	Number of samples	Percentage exceeding 0.1% individual phthalate limit
Hard	9	22.22 (2)
Soft	15	60 (9)
Total	24	45.83 (11)

False labelling of toys: Tests prove claims on labels are fraudulent

Type	Description	No of times individual 0.1% limit
Squeaky toy	Non toxic: Age 3-18 months	162
Inflatable	Age 2+	44
Teether	Food gradable silicone; designed for babies 6 months and older	3
Soft and hard biter	Designed for babies 6 months and older	2

Source: *Down To Earth*, January 15-31, 2010

HEALTH EFFECTS

Potential health effects	Phthalates associated
Pediatric exposure Preterm birth (shorter pregnancy duration), prenatal toxicity (antiandrogenic in fetal life)	DEHP & DINP
Respiratory effects Asthma and allergy in children Asthma allergy persistent wheezing, persistent cough, persistent phlegm, nasal congestion, respiratory infections	BBP & DEHP
Hepatic effects Increased liver weights, elevated liver enzyme levels, histological changes and in some cases tumors. Peroxisomal proliferation (a process related to metabolism of cholesterol and fatty acids)	DEHP & DINP
Developmental and reproductive effects Toxic to reproduction, capable of causing changes to both male and female reproductive systems in mammals, e.g. Development of testis in early life. Reduced testosterone, poor semen quality. Genital defects, reductions in anogenital distance in male infants. Premature breast developments in females.	BBP, DBP, DEHP, DMP, DEP & DNOP

Source: *Down To Earth,* January 15-31, 2010

metabolic, endocrine and reproductive systems are immature, they are more vulnerable (see Table: *Health effects*).

Phthalates have been shown to have pediatric exposure. Animal studies indicate that phthalates can cross the placenta and pass into breast milk, so prenatal exposure and exposure from breast-feeding may occur in humans. A recent study found measurable levels of nine phthalate metabolites in 163 infants who were born in 2000-2005. In 81 per cent of the infants, seven or more DEHP phthalate metabolites were above the limit of detection.

Infant exposure to lotions, powders, and shampoos were significantly associated with increased urinary concentrations of monoethyl phthalate, monomethyl phthalate, and monoisobutyl phthalate, and associations increased with the number of products used. Levels in the urine were closely correlated with use of infant care products (lotion, powder, shampoo) within 24 hours of urine sample collection, making it clear that what goes on a baby also goes into a baby.

A study on prenatal toxicity of three different dozes of DINP (0, 40, 200, 1000 mg/kg body weight with gestational days of five to 15 in rats) showed fetal effects (slightly increased rates of skeletal retardation and soft tissue and skeletal variations, occurrence of some soft tissue and skeletal malformations). These effects were seen with one of the types of DINP at maternal toxic doses (1,000 mg/kg body weight/day), but not at the two lower doses.

Another study investigated the possible role of DEHP exposure during pregnancy and a link between exposure to phthalates and pre-term birth. DEHP and mono ethyl hexyl phthalate (MEHP) levels were measured in cord blood of 84 consecutive births (77 per cent of the samples) at the Brindisi Hospital, Brindisi, Italy in 2003. Newborns that were MEHP positive had lower gestational age compared to MEHP negative infants. Findings suggest that exposure can begin *in utero* and that it may affect fetal development.

Phthalates are known to have respiratory effects as well. Exposure can trigger asthma and allergic symptoms in children. In a Swedish epidemiological study, exposure to phthalates – BBP, DEHP, and DBP – via house dust has been linked to asthma and allergic symptoms in children.

One of the phthalates, DEHP is a well-known hepatotoxin (toxic to liver) in rodents. Large amounts of specific phthalates fed to rodents have been shown to damage their liver and testes, and initial rodent studies also indicated hepato carcinogenity.

Recent studies have reported effects of phthalates on male reproductive health, such as reduced sperm production in laboratory animals. Phthalates may induce anti-androgenic or estrogenic effects in humans.

One of the first studies from Harvard School of Public Health linked phthalate exposure with harm to human reproductive health. Men who had monobutyl phthalate (MBP) or monobenzyl phthalate in their urine tended to have lower sperm counts, with the highest concentrations leading to the lowest sperm counts.

Developmental defects in male rat pups, similar to those seen in a syndrome termed testicular dysgenesis syndrome (TDS), have been documented after dosing pregnant rats with certain phthalates at high doses.

Studies have shown that there is a correlation between the phthalate metabolites in urine samples from pregnant women and a relatively short anogenital distance (AGD) in their male babies.

Studies have also indicated an association between plasticisers with known estrogenic and anti-androgenic activity and the cause of premature breast development in a human female population with exposure to DEHP.

5. *What are the substitutes for phthalates?*

Safer cost-effective alternatives exist, such as PVC-free toys that are manufactured without phthalates as well as phthalate-free plasticisers. The safer alternatives to PVC baby/children's products and

toys include toys made out of bio-based plastics, polyethylene, polypropylene, thermoplastic elastomers, and ethylene vinyl acetate (EVA) that are free of phthalates. These plastics do not require the use of phthalates since some are naturally softer; they pose fewer life cycle hazards because they are not chlorinated and do not release dioxins and furans during manufacture and disposal and are manufactured with chemicals that are less hazardous.

Another option is PVC with non-phthalate plasticisers. Danisco, a Danish company which is one of the largest manufacturers of food additives in the world, has introduced phthalate alternatives for toys and other products that have been approved for use in both the EU and the US.

Two of the most common alternatives to phthalates are acetyl tributyl citrate (ATBC) and 1,2-Cyclohexanedicarboxylic acid diisononyl ester (DINCH), which is derived from DINP and has a similar chemical structure. However, existing data on their chronic toxicity on humans is insufficient.

6. What are the global rules for phthalates in toys?

- ***European Union*** was the first to regulate the use of phthalates in toys. In 1999, it temporarily banned six phthalates used in childcare articles and toys made of soft PVC that can be put in the mouth by children under three. In 2005, it decided to restrict the use of three phthalates – DEHP, DBP and BBP – in all childcare articles and toys to 0.1 per cent concentration by mass of the plasticised material. Toys containing these chemicals in higher quantities cannot be sold in EU countries.

The EU proposed the same limit for three more phthalates – DINP, DIDP and DNOP (di-n-octyl phthalate) – but only in toys and childcare articles meant to be put in the mouth by children. Other toys were exempted from this restriction for want of more evidence of the toxicity of the three phthalates. The EU, however, noted that the three did pose a potential risk if used in toys. The restrictions came into force from January 16, 2007 and were to be reviewed by January 16, 2010. The EU was considering evidence that showed phthalates acting together can damage health in ways that each by itself would not.

EU regulations say products using phthalates do not have to mention their presence or carry a warning on the packaging. Only containers with more than 0.5 per cent of DBP, BBP or DEHP have to be labeled with the skull and crossbones symbol for the purpose of handling, according to the Phthalates Information Centre Europe, an industry body.

The EU also has a rapid alert system for non-food consumer products, under which member nations can access information about steps being taken by other member states and economic operators with regard to products posing a serious, long-term risk to health and safety of consumers.

- ***Denmark*** has gone a step ahead and placed a ban on the sale and import of toys and childcare articles meant to be put in the mouth that contain phthalates, not covered by EU regulations, at levels exceeding 0.05 per cent.

To ensure that toys available in the country are phthalate-free, the Danish government has also negotiated an agreement with retailers to voluntarily refrain from selling phthalate-containing toys meant to be put in the mouth between three and six years. Denmark taxes PVC and phthalate-containing products, domestic as well as imported.

- The ***US*** Congress enacted the Consumer Product Safety Improvement Act (CPSIA) in August 2008,

REPLACING PHTHALATES

Mattel, Hasbro and Toys "R" Us – US-based multinational companies – have announced they would meet the EU standards globally. Over the past four-five years, some of the United States' largest retailers including Wal-Mart, Target, Sears Holdings (Sears and Kmart), and Toys "R" Us have announced major policies to phase out or restrict toxic chemicals such as phthalates and/or PVC in children's toys and infant products.

Ban on phthalates in products that are intended for children exists in Europe, Argentina, Fiji and Mexico. Austria, Japan, Denmark, Finland, France, Germany, Greece, Norway and Sweden have banned the use of phthalates in manufacturing of soft vinyl toys.[1]

prescribing restrictions broadly similar to those in the EU on toys and childcare articles sold in US markets. The ban on DINP, DIDP and DNOP is *interim*. The Act stipulates two types of restrictions on phthalates.

The first part of the regulation, which came into force in February 2009, permanently bans manufacturing for sale, distribution and importing of children's toys and childcare articles containing more than 0.1 per cent of either DEHP, DBP or BBP. A toy is a product meant for a child of up to 12 years, while a childcare article refers to a product that a child of three years or younger uses for feeding, sleeping, sucking and teething.

The second part of the regulation seeks an interim ban on DINP, DIDP and DNOP (above 0.1 per cent) from being used in childcare articles or toys that can be placed in a child's mouth. By definition a toy or part of a toy can be placed in a child's mouth if in one dimension it is smaller than five centimeters. Toys that can only be licked are not covered under the regulation. The threshold levels are prescribed only for individual chemicals; no composite threshold is prescribed for more than one phthalate present in toys. The Act does not mandate labeling toys to indicate compliance with phthalate standards.

A seven-member Chronic Hazard Advisory Panel of scientists established under CPSIA will look at health effects of the full range of phthalates, individually and in combination, used in children's products. The panel had 18 months to complete its study – but there has been no update on its website since. After this the Consumer Product Safety Commission, tasked with implementing the Act, will evaluate the findings and consider banning products containing phthalates as hazardous.

The commission has devised detailed testing methods to identify the presence of phthalates. Under these methods the manufacturer is required to provide a certificate testifying its products have been tested for compliance with the commission's guidelines.

Since September 2009 the testing is specified to be done by an accredited third party laboratory. But the commission has stayed general certification until a panel of accredited labs is established. So the law is yet to be implemented effectively.

- ***China*** does not have standards for phthalates. According to the Toy Industry Association, China follows international standards dealing with safety aspects of toys related to mechanical and physical properties.

- The new phthalate requirement for toys under the ***Japan*** Food Sanitation Law (JFSL), which extends the scope to all plasticized material for six phthalates, became effective on September 6, 2011. The Japan Toy Association has aligned the requirement and test method for phthalates in the Japan Toy Safety Standard (JTSS) to be in accord with the Japan Food Sanitation Law. On August 23, 2011, the Japan Toy Association issued a notice announcing that the phthalate requirement will be revised in Japan Toy Safety Standard ST-2002 Part 3 by extending the scope to include all plasticized material for six phthalates.

7. How is India regulating phthalates?

Till April 2011, with regard to safety guidelines for toys, the Bureau of Indian Standards (BIS) had published three standards – none of which provided a limit for phthalates in children's toys and childcare articles:

- IS 9873 (Part 1): 2001/ISO 8124 – 1:2001 covers safety requirements of toys and safety aspects related to mechanical and physical properties
- IS 9873 (Part 2): 1999/ISO 8124 – 2:1994 covers safety requirements of toys and flammability requirements
- IS 9873 (Part 3): 1999/ISO 8124 – 3:1997 covers safety requirements of toys; migration of certain elements (has limits for heavy metals)

On April 20, 2011, the BIS released its draft regulations on standards on phthalates in toys,

following a Bombay High Court order on March 24, 2011. The court's direction was in response to a PIL filed by the Consumer Welfare Association in 2007 demanding stringent regulations for phthalates in toys.

In the draft regulations, the phthalate standards have been set at par with international norms. The draft regulates six phthalates commonly found in toys and childcare items – DEHP, DBP, BBP, DINP, DIDP and DNOP.

The draft states that DEHP, DBP and BBP will be regulated to be less than or equal to 0.1 per cent in all plastic toys and childcare products. Phthalates like DINP, DIDP and DNOP have been regulated to be less than 0.1 per cent, specifically for toys and childcare products for children aged four and below; these include toys like teethers.

The court had directed the Union ministry of commerce to issue a notification, once the BIS set the standards, mandating the phthalates standards in toys. The notification has not yet been done. Moreover, the BIS guideline with regard to toy production is self-regulatory and not mandatory. Toy manufacturers do not register for the ISI mark for their products and therefore, do not follow even the voluntary standard.

Indian toy companies are waiting for the notification on phthalates in toys to be issued. They are waiting for the government to help them in making the transition. The Indian Toy Manufacturers' Association wants the government to also import bulk raw materials that will make the production of toys safer. It says government should set a standard for all the industrial products that are used to make

THE INDIAN TOY INDUSTRY

The Indian toy industry has a turnover of about Rs 4,000 crore, of which Rs 1,500 crore is in the organised sector and Rs 2,500 crore the unorganised sector. The industry is characterised by small scale establishments and is labour-intensive. There are approximately 2,000 manufacturing units consisting of micro (1,500), small and medium (450) and large (20) units.

The demand for toys is growing at an annual rate of 20-25 per cent, while production is growing at 15-20 per cent. Indian manufacturers meet only 40 per cent of the domestic toy demand. State-wise, Delhi is the leading toy manufacturer with 44 per cent of the production in its kitty; Maharashtra follows with 17 per cent.[1]

the toys – this may make it easier to monitor the unorganised sector.

8. What did India do to stop import of toxic toys?

Phthalates were never regulated by the Indian authorities, though they did make a few botched attempts to regulate other safety aspects of toys like mechanical and chemical properties and the presence of certain heavy metals.

In January 2009, the authorities, under pressure from the judiciary, tried to regulate the quality of imported toys. To begin with, they banned the import of toys from China; then they issued a notification asking for all Chinese imports to conform to Indian standards. The scope of the notification was broadened later to cover toy imports from all countries.

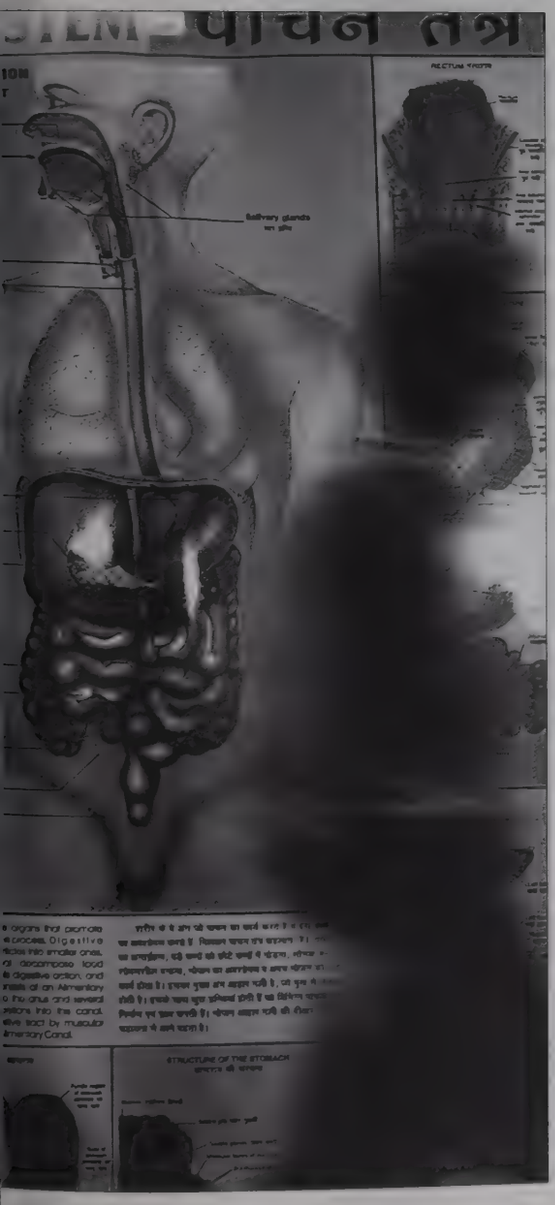
But while they had rules for imports, the domestic market remained unchecked. The regulation on imports expired on January 23, 2010. China went to the World Trade Organization with a complaint against India and India had to lift the ban on Chinese toys.

The BIS has drafted a set of rules for the presence of phthalates in toys, but these are yet to be notified. The toy market in this country cannot be regulated till the BIS rules are made mandatory for both domestic markets and imports. ●

FOUL FOOD

4

Health is rarely the topic of discussion at the United Nations General Assembly (UNGA). But in September 2011, the UN held a high level meeting to debate a strategy to tackle non-communicable diseases (NCDs). The problem was the rising numbers of the obese, which was making the world's people unfit and putting public health services under strain. The World Health Organization (WHO) had proposed targets to reduce NCD deaths, including a target for reduction in salt, fat and fructose corn syrup intake.



But it was too much to expect global leaders to indict the powerful processed food industry. So, a declaration was adopted calling for a multi-pronged campaign by governments, industry and civil society to curb the risk factors behind NCDs by 2013. NCDs are the biggest global killers today. Of the 57 million deaths that occurred globally in 2008, 36 million – almost two thirds (or 63 per cent) – were due to NCDs, comprising mainly cardiovascular diseases, cancers, diabetes and chronic lung diseases. But no deadlines were set. No clear roadmap was agreed upon.

There was also a consensus that marketing of unhealthy food to children must be regulated and checked. But the outcome on this matter had no clear commitments on how policies would be implemented to rein in junk food. There was no decision on salt and sugar reduction in processed food, no decision on making labeling easy to read.

There is convincing evidence that the consumption of high-energy foods, such as processed foods that are high in fats and sugars, promote obesity. At least 2.8 million people die each year as a result of being overweight or obese.

Now there is danger that obese people are at greater risk from exposure to toxins as well. This is a double whammy – bad food combined with bad environment.

4.1 JUNK FOOD

1. What is junk food?

There is no 'official' definition of junk food. But the medical dictionary variously defines junk food as "any food, which is low in essential nutrients and high in everything else — in particular calories and sodium. Junk foods are often highly salted like chips, high in refined carbohydrates (empty calories) like candy, soft drinks and high in saturated fats like cake and chocolates."[1]

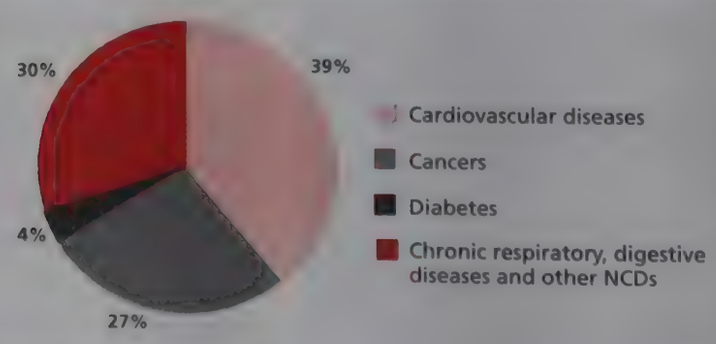
Most "junk food" falls into the categories of either "snack food" or "fast food", which are also defined as ready-to-eat food. The bottom line is this is food which is high on calories but low on nutrition. It should be eaten the least.

2. What is the danger of non-communicable diseases?

The World Health Organization (WHO) considers non-communicable diseases (NCDs) as the new global epidemic (see Graph: *Proportion of global NCD deaths under the age of 70*). Of the 57 million deaths that occurred globally in 2008, 36 million – almost two thirds (63 per cent) – were due to NCDs, comprising mainly cardiovascular diseases, cancers, diabetes and chronic lung diseases.

Worse, these diseases are preventable, says WHO. The causes are linked to lifestyle, food and environmental toxins. According to WHO, "These diseases are strongly associated and casually linked with four particular behaviors: tobacco use, physical inactivity, unhealthy diet and harmful use of alcohol. These behaviors lead to four key metabolic changes: raised blood pressure, obesity, hyperglycemia and

Proportion of global NCD deaths under the age of 70, by cause of death, 2008



Source: Anon 2010, *Global Status Report on Non-communicable Diseases*, World Health Organization, Geneva

hyperlipidemia. In terms of attributable deaths, the leading NCD risk factor globally is raised blood pressure (13 per cent of global deaths), followed by tobacco use (9 per cent), raised blood glucose (6 per cent), physical inactivity (6 per cent) and overweight and obesity (5 per cent)."[2]

The combined burden of these diseases is rising the fastest among lower-income countries, populations and communities, where they impose large, avoidable costs in human, social and economic terms. About one fourth of global NCD-related deaths take place before the age of 60.

3. How is diet related to disease?

The WHO says unhealthy diet is one key cause of the growing global burden of disease. Firstly, consumption of fruits and vegetables reduce the risk of cardiovascular diseases, and stomach and colorectal cancer. Secondly, and inversely, high salt consumption is an important determinant of high blood pressure and cardiovascular risk. In addition, high consumption of saturated fats and trans fatty acids is linked to heart disease. In this way, changing diets – low on nutrients and high on salt and fat – are directly indicted for ill-health.

Approximately 16 million (1.0 per cent) DALYs and 1.7 million (2.8 per cent) of deaths worldwide are attributable to low fruit and vegetable consumption. The WHO also adds, "There is convincing evidence that the consumption of high levels of high-energy foods, such as processed foods that are high in fats and sugars, promotes obesity compared to low-energy foods such as fruits and vegetables."[3]

4. What is the link between salt and disease?

The amount of dietary salt consumed is an important determinant of blood pressure levels and overall cardiovascular risk. The WHO recommends salt intake of less than 5 gram per person per day for the prevention of cardiovascular diseases. However, data from various countries indicates that most populations are consuming much more salt than this.

The WHO estimates that decreasing dietary salt intake from the current 9-12 gram per day to the recommended level of 5 gram per day would have a "major impact on reducing blood pressure and cardiovascular disease."

In Britain, for instance, there is clear evidence of how preventable this disease can be. In 1994, the government began its public health campaign to reduce salt in diets – demanding labeling of the amount of salt in food. In 2001, adults were taking 9.5 gram salt per day and the target was to reduce this to 6 gram by 2012. The prestigious medical journal *Lancet* reports that blood pressure among Britons has decreased. The government finds that it has prevented around 6,000 premature deaths annually by reducing salt by 10 per cent in processed food. It has saved US $2.5 billion in health costs.[4]

5. What is India's consumption of salt?

In India, there is no reliable estimate of the amount of salt consumed. An Indian Council of Medical Research (ICMR) survey done some two decades ago showed that adults consumed 13.8 gram of salt per day.

The National Institute of Nutrition's dietary guidelines suggest that India's current consumption is between from 5 to 30 gram per person per day across different states. Worse, nearly 40 per cent of the population consumes about 10 gram of salt per person per day. In other words, India has a serious salt problem.

6. What is the link between fat and trans fat consumption and disease?

The link between saturated fat and trans fat and increased risk of heart disease is well established. There is also evidence that the risk of type 2 diabetes is directly associated with consumption of saturated fat and trans fat and inversely associated with polyunsaturated fat from vegetable sources.

The WHO finds that there are huge variations across regions of the world in the amounts of fat available for human consumption. Energy from saturated fatty acids accounts for a third of the energy from total fat, with the notable exception of Southeast and South Asia, where it accounted for as high as 40 per cent of the total fat intake.

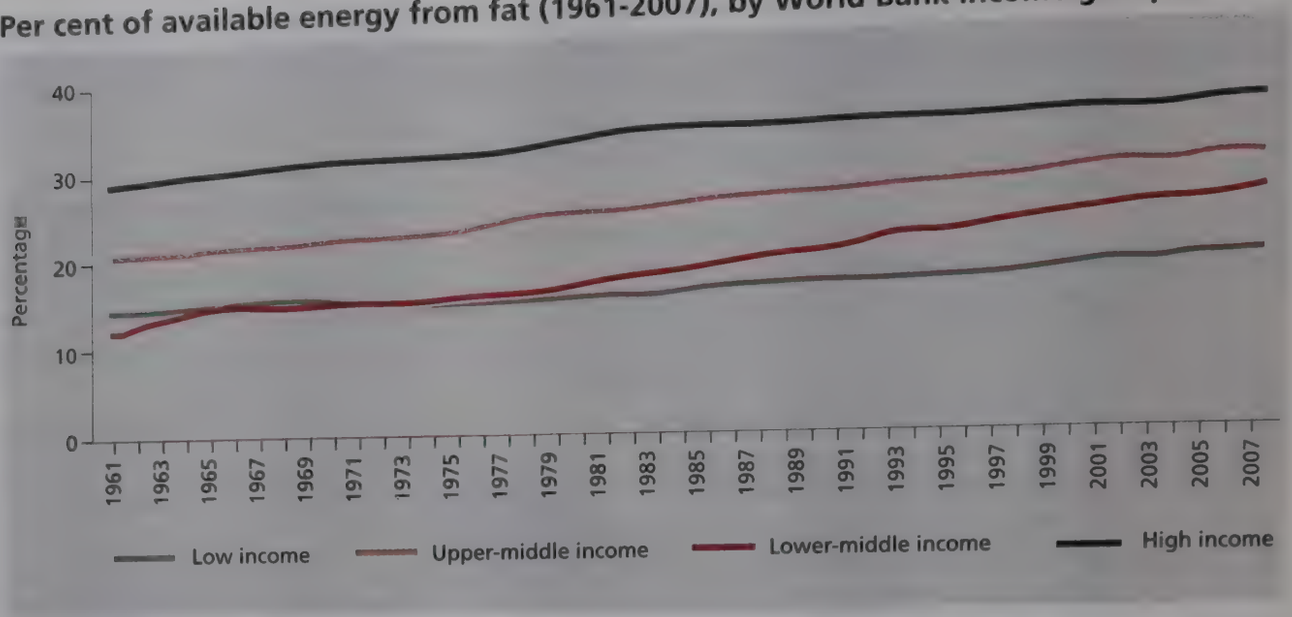
But what is clear is that the percentage of energy from fat is increasing across the world and across income groups. The World Bank estimates that the increase has been steady and rapid in lower-middle income countries (see Graph: *Per cent of available energy from fat*).

7. What is the link between sugar and disease?

Researchers are finding that increased sugar use is a common factor in all diseases of metabolic syndrome. Common sugar or sucrose consists of one molecule each of glucose and fructose. Glucose is the primary source of energy for the body, but is less than half as sweet as fructose. Being a primary energy source, glucose can be metabolised in every cell of the body, but fructose is broken down only in the liver.

A 2012 study published in *Nature* says high use of sugar – particularly fructose – is particularly harmful as it is addictive and induces us to eat more. In fact, the study claims that the toxic effects of

Per cent of available energy from fat (1961-2007), by World Bank income group, 2008



Source: Anon 2010, *Global Status Report on Non-communicable Diseases,* World Health Organization, Geneva

fructose are similar to that of alcohol: "Sugar has a clear potential of abuse. Like tobacco and alcohol, it acts on the brain to encourage subsequent intake. There are numerous studies examining the dependence producing properties of sugar in humans. Specifically, sugar dampens the suppression of the hormone ghrelin, which signals hunger to the brain. It also interferes with the normal transport and signaling of the hormone liptin, which helps produce a feeling of satiety, and it reduces dopamine signaling in the brain's reward centre, which in turn reduces pleasure derived from food, compelling individuals to eat more (see Table: *Deadly effect...*)."

So sugar, say researchers, is an empty calorie, but there is nothing empty about these calories: "A growing body of scientific evidence shows that fructose can trigger processes that lead to liver toxicity and a host of other chronic diseases."[5]

The authors suggest that as in the case of tobacco and alcohol, taxation would be the best way to regulate abuse of sugar. The tax would be added to processed foods that contain any form of added sugar – from carbonated drinks to sugared cereal.

Global sugar supply (in the form of sugar and sugar crops, excluding fruit and wine) expressed as calories per person per day, 2007

Source: Food and Agriculture Organization (FAO)

DEADLY EFFECT

Chronic ethanol exposure	Chronic fructose exposure
Haematological disorders	
Electrolyte abnormalities	
Hypertension	Hypertension (uric acid)
Cardiac dilatation	
Cardiomyopathy	Myocardial infarction (dyslipidaemia, insulin resistance)
Dyslipidaemia	Dyslipidaemia (de novo lipogenesis)
Pancreatitis	Pancreatitis (hypertriglyceridaemia)
Obesity (insulin resistance)	Obesity (insulin resistance)
Malnutrition	Malnutrition (obesity)
Hepatic dysfunction (alcoholic steatohepatitis)	Hepatic dysfunction (non-alcoholic steatohepatitis)
Fetal alcohol syndrome	
Addiction	Habituation, if not addiction

Source: R H Lustig 2010, *Journal of the American Dietetic Association*, 110, 1307–1321.

The US, which consumes an average of 216 litre of 'soda' per year per person, of which 58 per cent is sugar, is considering a small tax which will raise prices by about 10-12 US cents. However, this would be too small to reduce consumption: doubling the price could make a difference.

But ultimately, "food producers and distributors must reduce the amount of sugar added to foods. But sugar is cheap, sugar tastes good and sugar sells". The US Food and Drug Administration could set the table for change by removing fructose from the 'Generally Regarded as Safe' (GRAS) list, which allows manufacturers to add unlimited amounts to any food. Clearly, the added sugar problem is here to stay and it is neither sweet nor easy.

But this is not all: the industrial process of making sugar sweeter is even worse. Called high-fructose corn syrup and high-fructose maize syrup, this enzymatic process takes plant syrups and converts some of its glucose into fructose to produce the desired sweetness. So, while sucrose is composed of equal amounts of the two simple sugars – 50 per cent fructose and 50 per cent glucose – the typical high-fructose corn contains a minimum of over 55 per cent fructose and 42 per cent glucose. Larger sugar molecules called higher saccharides make up the remaining 3 per cent of the high-fructose corn syrup.

As a result of the manufacturing process of high-fructose corn syrup, the fructose molecules in the sweetener are free and unbound, ready for absorption and utilization. In contrast, every fructose molecule in sugar that comes from cane or beet is bound to a corresponding glucose molecule and must go through an extra metabolic step before it can be utilized.

In the US, consumer foods and products typically use high-fructose corn syrup as a sweetener. It is this that makes our processed food so sweet and desirable and not so healthy, say food researchers. The industry, however, disputes this view. In 2010, a Princeton University research team demonstrated that all sweeteners are not equal when it comes to weight gain: rats with access to high-fructose corn syrup gained significantly more weight than those with access to table sugar, even when their overall caloric intake was the same. This is because excess fructose is metabolised to produce fat, while glucose is largely processed for energy or stored as a carbohydrate, called glycogen, in the liver and muscles.[6]

Now there is more news about why sweetened beverages – everything from colas to other such drinks – are bad news for obesity. A 2012 study published in the *American Journal of Clinical Nutrition* concludes that replacing water and removing sugary drinks (two servings) in diets have led to a 2-kg weight loss in six months. In the US, the percentage of kilocalories coming from beverages has almost doubled between 1965 and 2002 – reaching as much as 21 per cent. The problem is sugar. Sweetened drinks should be cut off our diets to stay healthy – this is the message.[7]

8. What is the 'fat' problem in the world?

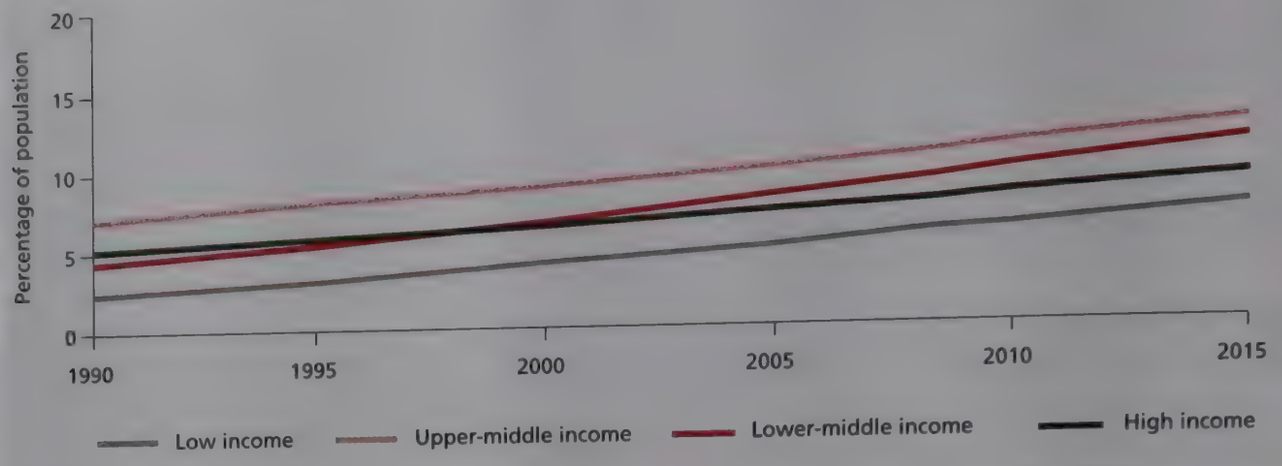
The WHO estimates that worldwide, 2.8 million people die each year as a result of being overweight and obese. Risks of heart disease, stroke and type 2 diabetes, and cancer increase steadily with increasing body mass index (BMI) – a measure of weight relative to height.

According to WHO, the goal for individuals should be to maintain a BMI in the range of 18.5-24.9 kg/m^2.

In 2008, 35 per cent of adults aged 20 years and older were overweight. But more dangerously, the prevalence of obesity – when populations are so overweight that they are defined as obese – had nearly doubled between 1980 and 2008.

Even more worrying is that children are increasingly losing this battle of the bulge. Estimates indicate that in 2008 as much as 6 per cent of the world's infants and young children were overweight. The highest prevalence is in upper-middle income group, but the fastest rise is in lower-middle income group (see Graph: *Infant and young child overweight trends from 1990 to 2015*).

Infant and young child overweight trends (1990 to 2015), by World Bank income group



Source: Anon 2010, *Global Status Report on Non-communicable Diseases*, World Health Organization, Geneva

9. But why should poor India worry about a rich man's disease?

The Public Health Foundation of India has recently published a report compiling the dangers of non-communicable diseases in India. According to this report, in 2005, NCDs account for as much as 53 per cent of the total mortality and 44 per cent of the disability adjusted life years in India. And by 2030 this percentage is likely to rise to 67 per cent of total mortality (see Graph: *Cause-specific mortality in India*).[8]

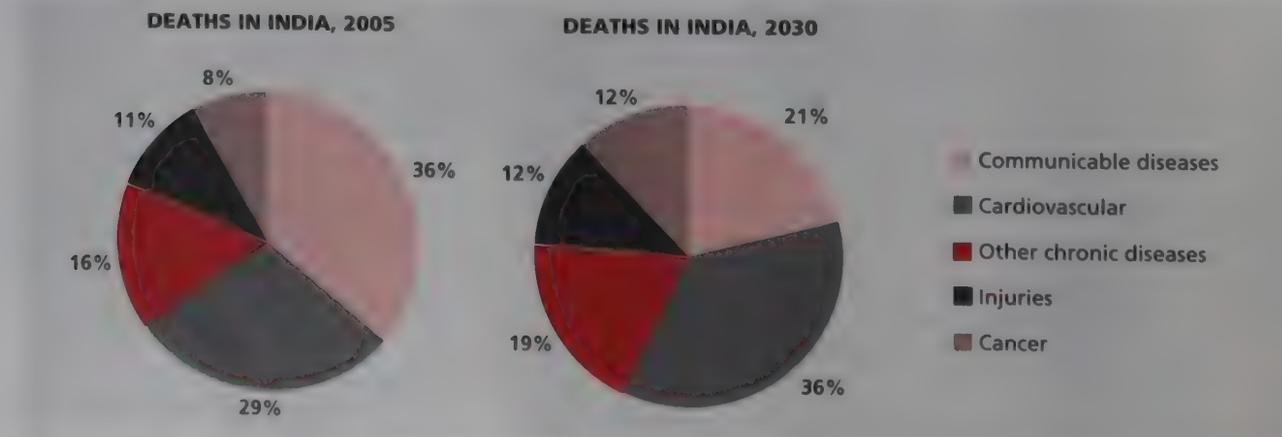
India is also no different from the rest of the world in terms of NCD risks – blood pressure tops the cause of death and overweight and obesity add to the burden (see Graph: *Deaths caused by nine leading NCD risk factors in India*).

This data is confirmed by the Integrated Disease Surveillance Project report on non-communicable disease risk factors, for seven states of India. The report, by the National Institute of Medical Statistics, reveals that there is a shocking increase in hypertension, not just in urban India but also in rural India.[9] The reasons are clear – rural India is getting urbanised in terms of food and lifestyle with use of tobacco, alcohol and junk food on the one hand, and lack of exercise and decreased consumption of vegetables on the other hand.

10. Why is India the diabetes capital of the world?

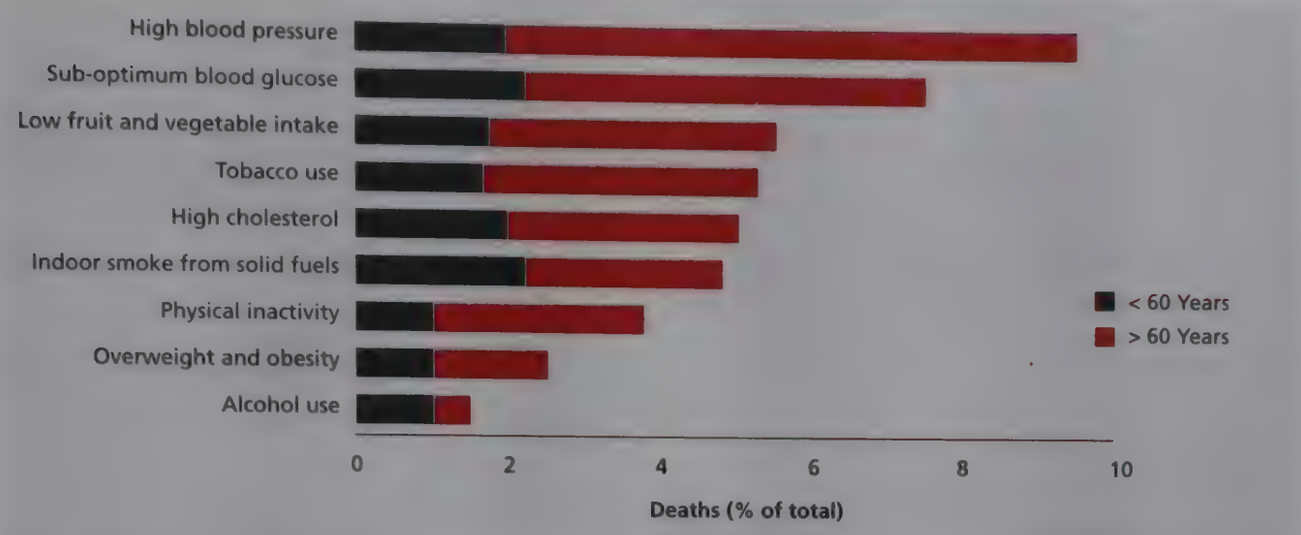
It is now understood that for genetic reasons, compounded by food and lifestyle, India is well on the way to be the diabetes capital of the world. Currently, it is said that more than 7 per cent of Indians have diabetes, which is at par with the incidence in a richer Australia and higher than that in the UK. Worse, it is increasing rapidly – diabetes incidence in the country is expected to increase by 150 per cent over the next 15 years. Already, 50 million Indians are known to suffer from this rich person's disease, which impairs the immune system

Cause-specific mortality in India



Source: Sailesh Mohan, K Srinath Reddy and D Prabhakaran 2011, *Chronic Non-Communicable Diseases in India: Reversing the tide*, Public Health Foundation of India, New Delhi

Deaths caused by nine leading NCD risk factors in India (in per cent)



Source: Sailesh Mohan, K Srinath Reddy and D Prabhakaran 2011, *Chronic Non-Communicable Diseases in India: Reversing the tide*, Public Health Foundation of India, New Delhi

and adds to other afflictions.

Younger Indians are now falling prey to diabetes. According to doctors and researchers, the average age of onset of diabetes in Indians is a decade earlier than other races. This means that younger Indians are at a greater risk from cardiac problems as well.[10]

11. How 'fat' is India?

India has the dubious distinction of a country with a twin burden – under nutrition and over nutrition. In India, 14.8 per cent of women and 12.1 per cent of men are obese or overweight. This is according to the third National Family Health Survey (NFHS)-3 data (2005-06). No data for obese men is available from the last survey conducted in 1998-99, but the data available for women shows a 4.2 per cent increase in obesity since 1998-99[11].

But what is worrying is that close to 30 per cent women in urban India are obese and that this problem is the worst with educated women (see Table: *Whither education?*).

However, other studies indicate a growing bulge. The Jaipur Heart Watch studies show increasing trend in overweight/obese urban men – from 21 per cent in 1994 to 51 per cent in 2005. Women are gaining weight even faster – going from 16 per cent in 1994 to 58 per cent in 2005.

There is no national study on childhood obesity, but local studies suggest a growing and deadly weight problem in this vulnerable group.

12. What is the problem of obesity and disease in Indian children?

Research indicates that 77 per cent of obese children in India would end up as obese adults. While it is well known that insulin resistance and diabetes is particularly high in adult Asians, the question is how does it affect children, and more importantly, does childhood obesity hurt us even as we grow older? Studies say it does. Overweight and obese children are at substantial risk of developing multiple cardiovascular problems, say doctors. Doctor and researcher Anoop Misra explains that hyperinsulinemia and insulin resistance are biomarkers which predict cardiovascular risk in adulthood.[12]

13. Why should India worry? Do we eat junk food?

According to the National Restaurant Association of India (NRAI) 2010 report, the fast food industry in India is currently estimated to be worth between Rs 6,750-Rs 8,000 crore, growing at a compound annual

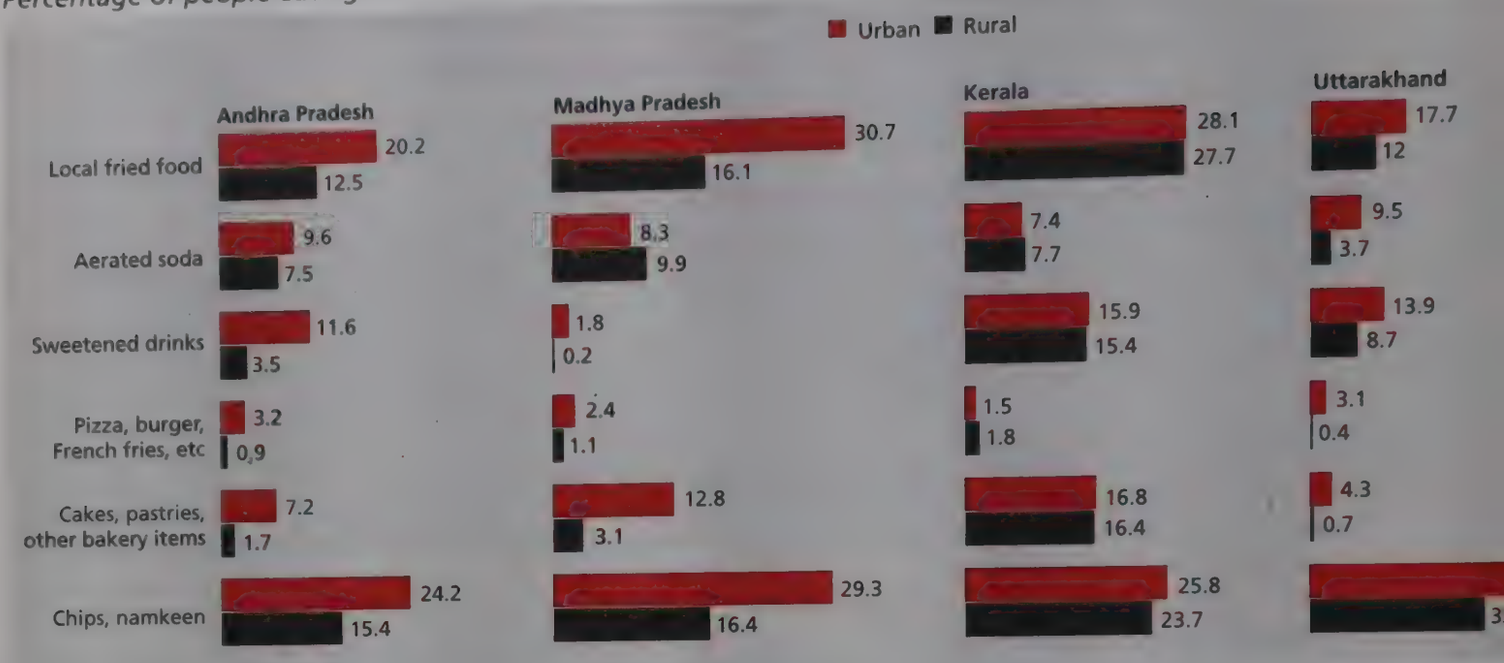
WHITHER EDUCATION?

Key Indicators for NFHS-3	NFHS-3 (2005-06)	Urban	Rural	No Education	<5 years complete	5-9 years complete	Above 10 years complete
Women who are obese or overweight	14.8	28.9	8.6	7.6	12.1	18.7	30.7
Men who are obese or overweight	12.1	22.2	7.3	3.9	6.3	10.2	23.6

Note: All figures are in percentage
Source: National Family Health Survey (NFHS-3), 2005–06, Volume I

The junk that we eat

Percentage of people eating fast food once a week



Low on healthy diet

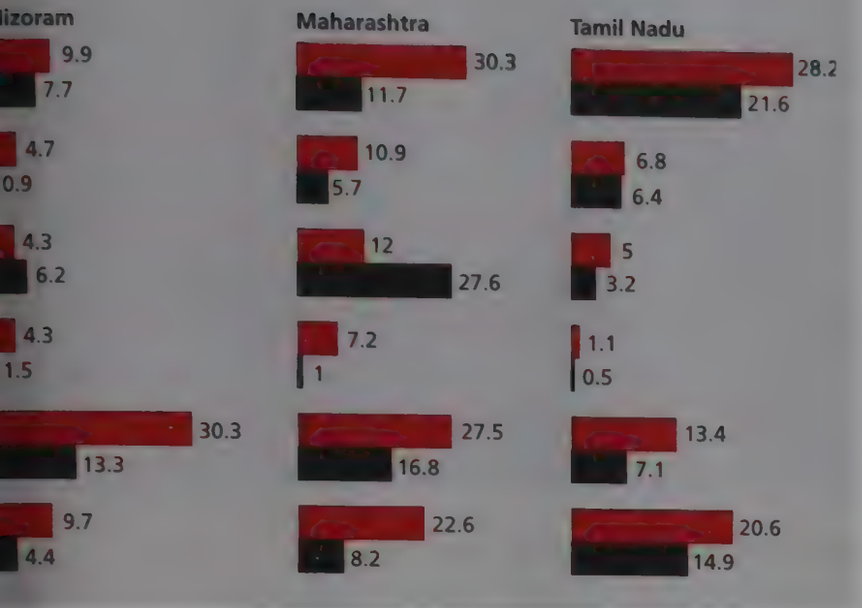
Number of days people eat fruits and vegetables in a week

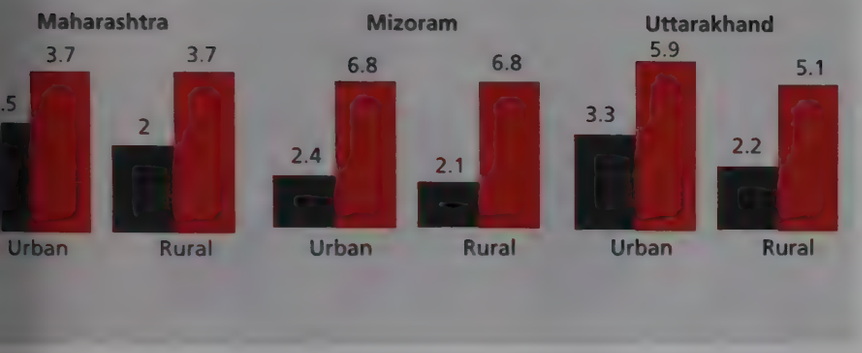
Source: NIMS 2009, Integrated Disease Surveillance Project, Non-Communicable Disease Risk Fact Survey, National Institute of Medical Statistics, New Delhi

growth rate of 35-40 per cent. A major chunk of these markets is ruled by global players like McDonald's, Yum! Brands (Kentucky Fried Chicken, Pizza Hut), Domino's, Subway, Taco Bell, Coca Cola and Barista but domestic players are not lagging behind. Nirula's, Pizza Corner, Coffee Day Group, Haldiram and Bikanervala capture a fair share of the fast food segment.

The Integrated Disease Surveillance Report, which found that rural India was fast catching up with modern diseases, also noted that people were eating less fruits and vegetables and more fast food. It found that across the seven states in which it conducted its study, in urban and rural areas, there was a growing and substantial percentage of people eating fast food once a week.

As much as 10 per cent of the urban population drank aerated sodas at least once a week and as much as a quarter admitted to eating chips and





namkeen at least once a week (see Graphs: *The junk that we eat* and *Low on healthy diet*).[13]

It is clear that the modern lifestyle is catching up with us.

14. Why is 'fat' India even more at risk?

A few years ago, doctors realised that Indians are at higher risk because our body mass index (BMI) is lower than Caucasian populations. This is because when the normal limits of BMI were declared in the 1980s, it was done based on a survey conducted with 80,000 Caucasians. Researchers from Europe and the US correlated the BMI to mortality rates.

This survey defined the term 'obesity' – those with BMI between 25- 29.9 kg/m^2 were overweight while those with a BMI of more than 29.9 kg/m^2 were obese. The International Task Force on Obesity and the WHO accepted these limits, making them universal.

However, Asian doctors questioned the survey as it was based on Caucasians. In the year 2000, Dr Anoop Misra, then a professor at the department of internal medicine, All India Institute of Medical Sciences, New Delhi, observed a rise in the number of diabetics although they couldn't be identified as obese.

Universally, BMI is measured by two rules: body fat as a standard and comparing the BMI against it and by comparing BMI against diseases. Misra found that Indians had higher body fat even at a lower BMI. For instance, at a BMI of 25-26 kg/m^2, 10 per cent of Caucasians had diabetes; compared to that, 10 per cent of Indians had diabetes at a BMI of 22 kg/m^2.

Indians become classifiable as overweight or obese at a lower BMI and this is due to the genetic makeup. Indians have less muscle content and more fat content when compared to Caucasians. And with a change in diets, that become increasingly laden with saturated fats, more carbohydrates and less fibre – a diet that goes with a more "westernised" lifestyle, meaning one that includes eating more meat, eating out regularly and eating more 'fast' or 'junk' food – Indians gain fat really fast.

In 2008, Dr Anoop Misra and a group of 100 doctors from India studied parameters like body fat and risk of diseases and announced new guidelines at a meeting held in New Delhi on November 15-16, 2008.[14]

The WHO has accepted this changed guideline for Asian Indians. Now, as compared to the rest of the world, healthy Indians must have a lower BMI. While the upper range of BMI for a normal adult is 24.99 kg/m^2, Asian Indians should have a BMI up to 22.99 kg/m^2.

Indian also have a central obesity problem – we are genetically prone to put on fat around our abdomen. This is an important measure of obesity and cardiometabolic risk and so health professionals have been working to re-define the healthy waste size for Indians. The optimal waist circumference is reported to be 85-87 cm for men and 80-82 cm for women.[15]

15. Do children know what they are eating is unhealthy?

No. In fact, children know very little about their diet and its implications for their health, say researchers. Worse, mothers know as little (see Tables and Graphs on page 88). This, say doctors, is the one big reason for the 'fat' problem – little knowledge and even less concern. Children also say that interesting advertisements inspire them to purchase junk food: consumption of so-called 'lifestyle foods' among children is on the increase. When doctors S Gulati

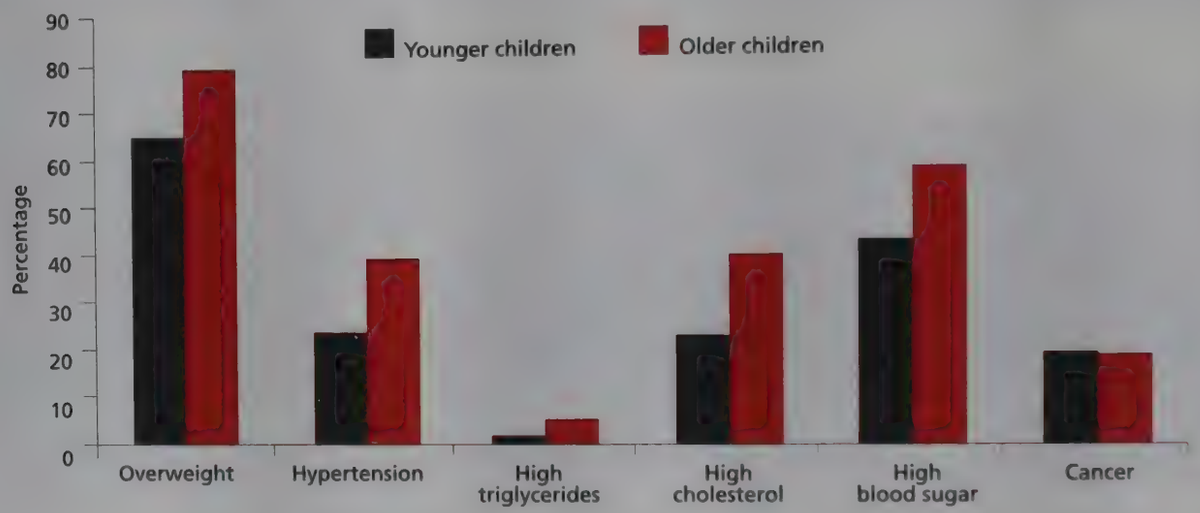
CONSUMPTION OF LIFESTYLE FOODS AMONG MOTHERS

Item	> 2-3 times a week	
	Mothers of younger children	Mothers of older children
Chips / Pringle	53	57
Popcorn	29	36
Soft drinks	25	28
Ice creams	43	48
Cakes / muffins	24	29
Chocolates	47	57
Fried food	39	38
Fast food	15	15

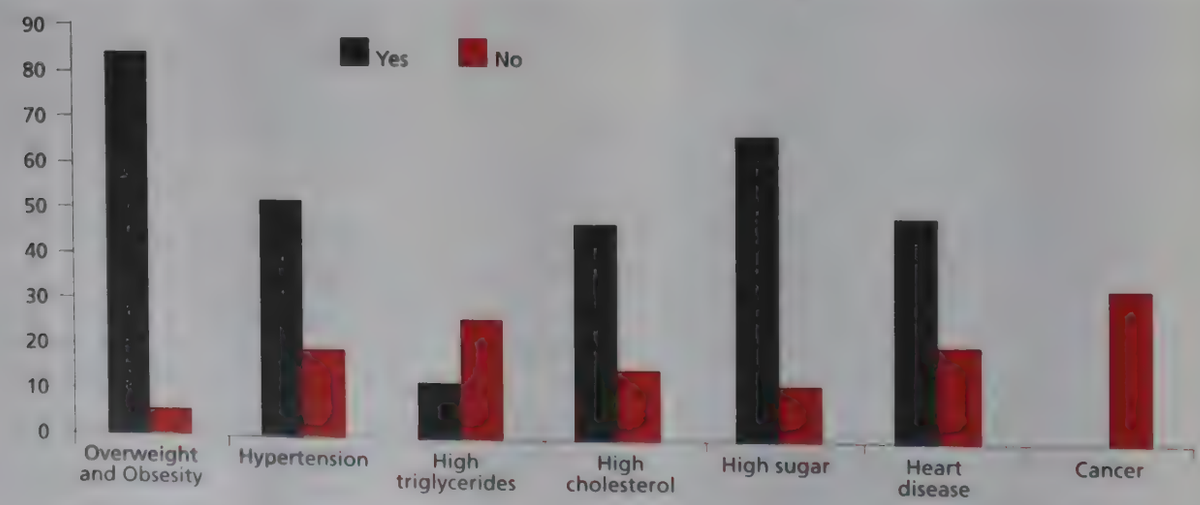
CONSUMPTION OF LIFESTYLE FOODS AMONG CHILDREN

Item	> 2-3 times a week	
	Younger children	Older children
Chips / Pringle	68	66
Popcorn	33	28
Soft drinks	30	41
Ice creams	44	58
Cakes / muffins	35	32
Chocolates	71	75
Deep fried food	40	57
Fast food (pizza/burger)	22	31

Knowledge regarding unhealthy diet and diseases among children

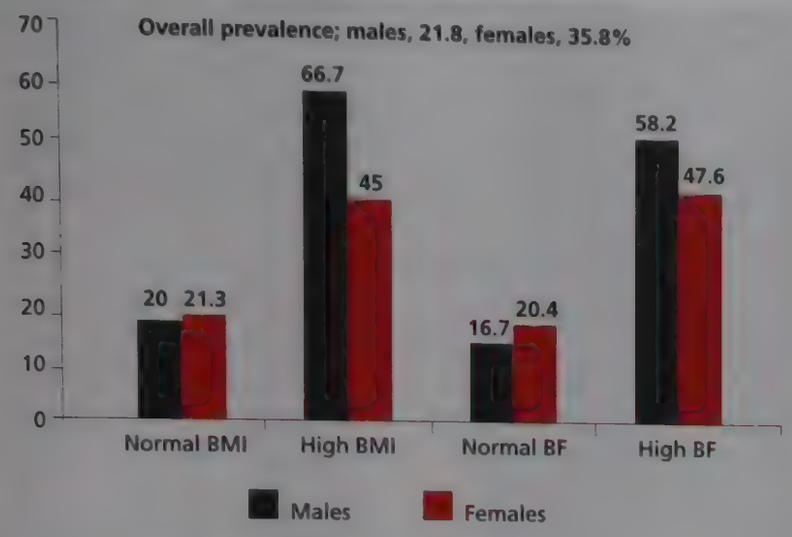


Knowledge regarding unhealthy diet and diseases among mothers



Source for tables and bar graphs on this page: Anoop Misra and Seema Gulati 2010, unpublished data, presented at CSE's South Asian Media Briefing on Food Safety and Environmental Toxins, March 29-30, 2012, New Delhi

Prevalence of insulin resistance (by Homa-IR) in post-pubertal Asian Indian children (n=396)



Source: Anoop Misra *et al* 2004, *International Journal of Obesity*

and Anoop Misra collected data on eating habits of Delhi children, they were shocked. Between 60-70 per cent of children in different age groups consumed chips at least two-three times a week; the mothers were just as bad. Children are eating bad and exercising less. Studies done in Delhi point to the growing sedentary habits in children, who spend most of their time watching TV, using computers or playing video games.

16. *What makes 'junk' food so deadly?*

Junk food is popular because it is tasty. But it is unhealthy – it is low in fiber, high in fat, and high in sugar in liquid form. Studies have shown that despite being unhealthy, junk food induces gorging that leads to obesity.

Published in 2009 in *The Journal of Clinical Investigation*, a study has shown that the fat from fatty acids can affect the brain. The study, by Deborah Clegg, assistant professor of internal medicine at UT Southwestern, suggested that when we eat something high in fat, the brain gets 'hit' with the fatty acids, and the fat molecules cause the brain to send messages to the body's cells, warning them to ignore the appetite-suppressing signals from leptin and insulin, hormones that are involved in weight regulation. And since the body does not get the signal that it is satiated, it leads to over eating[16].

Another study published in *Neurology* shows that eating too much junk food or food rich in trans fats can shrink the brain similar to that associated with Alzheimer's disease. The study also showed that if the diet was rich in vitamin B, C, D and E and high in omega-3 fatty acids, the brain benefitted from it.

Though this study was done on a set of people above 65 years, it is known that the impact of trans fatty acids begins to take place at the fetal stage[17].

Adverse fatty acid supplies during fetal and child development can alter fatty acid composition of membrane phospholipids (these are the lipid/fatty cells in the membrane and have an impact on the fattiness of the child) and storage triglycerides (they store unused calories in humans, and are an important measure for the health of the heart: the lower the better). This can lead to a disruption in cellular environments, structure and function.

Avoiding hydrogenated fats is also important for the growing brains of children as it can lead to attention deficit hyperactive disorder (this is a psychiatric disorder mainly in children and is characterised by a coexistence of both attention problem and hyperactivity).

Studies also show that too much of junk food alters the chemistry of the brain and is addictive like cocaine. High-fructose corn syrup (HFCS), monosodium glutamate (MSG), hydrogenated oils, refined salt, and various other chemical preservatives found in processed junk food do the same thing to a person's brain as cocaine does[18].

Another study conducted by researchers at both the University of Texas in Austin (UT) and the Oregon Research Institute found that prolonged consumption of junk foods results in reduced activity in the striatum, a section of the forebrain that registers reward.

In other words, just like with drugs, those addicted to junk food require ever-increasing amounts of it to get the same "high." In a correlative study, researchers identified a similarity in dopamine production levels between drug addicts and junk food addicts. Addiction to either one essentially causes the brain receptors that receive dopamine signals to lose their responsiveness. As a result, addicts require increasing amounts of the addictive substance to receive the same level of satisfaction.

Studies have also shown that as early as the age of 30, arteries could begin clogging and lay the groundwork for future heart attacks. What children eat from puberty affects their risks of prostate and breast cancer. Osteoporosis and hypertension are other diseases that appear to have their earliest roots in childhood when lifelong eating habits are being formed.

Children are especially vulnerable. Poor diets can slow growth, decay new teeth, promote obesity and sow the seeds of infirmity and debilitating disease that ultimately lead to incurable illnesses. Food containing low nutrition value tends to reduce the IQ level of children (see Box: *Catching them young*).

Most of the times these junk foods are laced with

CATCHING THEM YOUNG

Children and young people are the food industry's best customers. Hook them to a brand and you have had them for life. In USA, the number of overweight children has doubled and number of overweight adolescents has trebled since 1980. Obesity is now the most prevalent disease among children and young adults in the US. Experts blame poor eating habits and lack of physical exercise for this.

In 1999, companies spent an estimated US $30 billion on direct and indirect food advertising. A mint brand could have an advertising budget of US $10 million, but a US government campaign like the 'five a day fruit and vegetable programme' in its best funded year had a US $2 million budget, says a World Bank study. For every dollar spent in the US on nutrition education, the food industry spends an estimated US $5,000 on food advertising.

Coca-Cola, the world's largest soft drink group, won the global marketing rights for the first Harry Potter film at a reported cost of US $182 million. Millions were spent on publicity work. Studies show that one in three North American children now eat at a fast food restaurant every day. Soft drink consumption in the US has increased by 300 per cent in 20 years, and serving size per individual has increased from 6.5 oz (192 ml) in the 1950s to 12 oz (354 ml) in the 1960s and 20 oz (591 ml) by the late 1990s.

Even schools aren't safe from soft drinks and fast food. They sign "pouring rights" with soft drink companies to get funds. Approximately 60 per cent of middle and high schools in the US have soda vending machines and approximately 10 per cent of these schools have long-term contracts with soda companies.

The vending machines are put up at vantage points in schools; even water fountains have been removed to create space for them. Such contracts have provided schools with more than US $200 million in revenue, but the consequence is that 56 per cent to 85 per cent of school students consume at least one soft drink daily.

artificial colors, which are often inedible, carcinogenic and harmful to the body. These foods and their colors can affect digestive systems, the effects of it emerging after many years. Studies have found that food coloring can cause hyperactivity and lapses of concentration in children.

Doctors at Harvard University and the University of Murcia, Spain, have found that junk food can make young men infertile, even if they're physically fit and in good health. Those who ate high amounts of junk food, such as fried foods, processed red meats and potato chips were found to have sperm of poorer quality than their counterparts who ate a nutritious diet high in whole grains, vegetables and fish. In particular, participants who consumed high levels of trans fats, naturally found in fried foods, had the worst quality sperm – that is, sperm that was least likely to survive the journey to fertilize an egg. This was the case even for men who had a healthy weight and exercised regularly.

17. What are the global politics of regulating for good food?

In 2002, the WHO began discussions on global regulations on obesity, suggesting the need for a global agreement to cut salt, sugar and fat and put bans on advertising for bad food, at least during children's programmes. Its technical report, TRS 916, set out the agenda clearly – stating that the calorie from sugar must be restricted to 10 per cent of the daily diet. The report indicted the powerful food processing industry and its aggressive marketing and advertising blitz for selling 'bad' food.

The WHO went on to propose a global strategy against obesity – advocating for subsidy on healthy food and taxes and ban on what is unhealthy. Under the proposed strategy, governments would have to come up with national dietary guidelines, correct and scientific information on food and nutrition labeling, and incentivise the marketing and promotion of good food. They would also have to implement strategies to make unhealthy food more expensive and remove subsidies on sugar. It also called for the food industry to limit the levels of saturated fats, trans fatty acids, free sugar and salt in food products.

This proposed strategy riled the world's food industry. The US corn refiners association, snack food association and sugar association stepped up their lobbying against the strategy, calling it "scientifically flawed and misguided". In January 2004, just before the WHO health assembly, the US government formally condemned the report and its findings: "There is unsubstantiated focus on good and bad foods and a conclusion that specific foods are linked to non-communicable disease and obesity." The International Sugar Organisation argued that the strategy was bad as it 'demonised' its product, was unnecessary and flawed. Even India succumbed to the pressure from the food industry and took a contrary position. In March 2004, its official communication to WHO said, "There is disproportionate focus on energy-rich diets, which is a narrower issue more relevant to developed countries."[19]

At the 57th World Health Assembly in May 2004, the global strategy on diet, physical activity and health was endorsed. But the final agreement was watered down and weak – leaving governments to do as best as possible, but setting no targets. Industry had won this round. It was agreed that based on this strategy, countries would come up with their plans by May 2006. Since then, some governments, particularly in the already industrialised world, have acted (see Question: What are countries doing to ban junk food?). But many countries, particularly in the developing world, have not. In the meantime, the processed food industry has moved into these countries with aggressive marketing leading to a growing health burden.

In 2011, the matter was raised at the UN General Assembly – a special session was called in New York on September 19-20 to declare non-communicable diseases a global priority. This was only the second time that the UN assembly was convened on a health issue – the first one was on HIV/AIDS. But industry had already been working overtime to dilute the recommendations. A Coca-Cola representative chaired the crucial working group on food and drink. Not surprisingly, the group suggested that there should be no global agreement – only voluntary self-regulation by industry.[20]

With this obvious pressure from global industry, the UN General Assembly adopted a watered down political declaration to reduce the burden of chronic lifestyle diseases. It only calls for a comprehensive campaign by all to combat this global problem, but sets no targets on how the diseases will be curbed. This, when the draft had set clear global goals: 25 per cent reduction in deaths from NCDs, 40 per cent reduction in tobacco smoking and reduction in salt intake. Margaret Chan, director general of the WHO, called these diseases 'slow-motion disaster". Governments, however, have refused to take stern steps to deal with this new and deadly global epidemic.[21]

18. What are countries doing to ban junk food?

In spite of all efforts, there is no global obesity protocol. But countries are working individually to fight the battle of the bulge by introducing

restrictions and bans on 'junk food'.

On January 21, 2011, the WHO formally issued a recommendation asking for a ban on junk food in schools and playgrounds in order to promote healthy diet and tackle child obesity[22]. "Settings where children gather should be free from all forms of marketing of foods high in saturated fats, trans fatty acids, free sugars or salt," said the WHO. "Such settings include, but are not limited to, nurseries, schools, school grounds and pre-school centers, playgrounds, family and child clinics and pediatric services and during any sporting and cultural activities that are held on these premises," it added.

It also said that "children throughout the world are exposed to marketing of foods high in fat, sugar or salt, which increases the potential of younger generations developing non-communicable diseases during their lives". But critics say the WHO did not go far enough by not asking governments to ban advertising directed at children for foods high in saturated fats, sugars or salt.

Some countries are beginning to act.

Britain has the biggest obesity problem in Europe, with nearly 26 per cent of the population being obese[23]. According to the National Obesity Forum, a group working on raising awareness on the growing impact of obesity, it is estimated that if steps are not taken, then by 2050, nearly half of the British population will become obese or overweight, affecting not just the national health services but also the workforce[24].

The country banned junk food in schools in 2005[25]. A ban on junk food ads during television programs aimed at children below 16 years came into force in August 2008[26].

But corporate pressure is high against this move. The UK Food Safety Agency, in 2010, came under a lot of pressure for pushing a Europe-wide 'traffic light system' that required food companies to label the front of their products with red, amber or green symbols to denote the amounts of fat, saturated fat, salt and sugar contained in each serving. There were rumours that the country's Food Standards Agency (FSA) would be dismantled in July 2010; but this was opposed and checked. UK consumer advocates are now calling for their government to explore provisions allowed under the new EU regulation on food information to make sure that the 'traffic light labelling' is used on all food products[27]. Currently, only a handful of supermarkets follow this labeling system.

Scotland, in 2012, has proposed to ban advertisements of junk foods in television shows aired before 9 pm[28].

The US, the Mecca of bad food, has now decided to take action against its obesity epidemic. In 2010, the US government proposed a law to ban junk food in

schools, a decision that still remains to be taken[29]. Meanwhile, elementary schools in Arizona, Georgia, Kentucky, Louisiana, Maryland, Mississippi, Nebraska, New Jersey, New York, and West Virginia have already banned the sale of junk food in schools until at least after lunch[30].

Mexico has banned junk food in all its public, private and elementary schools in May 2010. This was a part of its nationwide anti-obesity campaign[31].

The United Arab Emirates banned junk food and soft drinks in all its schools in Abu Dhabi in 2010[32].

In Canada, the Ontario state government banned candies, chocolates, fries, pops and energy drinks in school premises in September 2010[33].

Denmark imposed a fat tax on junk food in October 2011. The surcharge will be levied on food items like butter, milk, cheese, pizza, meat, bacon, ice cream and processed food if they contain more than 2.3 per cent saturated fat. According to the new taxes imposed, the consumers will have to shell out about 20 per cent more for a packet of butter and a little more than half a Krone (1 Krone = 01.7 US $) for a packet of chips [34]. The government hopes that by imposing such taxes on junk food they will be able to fund the increased health care costs of treating the obese population. Also, high prices will dissuade people from eating food high on saturated fat[35].

Hungary had imposed tax on food that was high in sugar, fat, carbohydrates and salt in September 2011. A tax was also imposed on carbonated beverages, alcohol and drinks with high caffeine levels such as energy drinks[36].

Apart from Hungary and Denmark, other countries in Europe too have experimented with similar taxes. Switzerland, Austria and Denmark have already banned trans fats, while Finland and Romania are considering fat taxes[37]. Brazil, Mexico and Taiwan too are working towards getting a fat tax in place.

19. What is India doing about junk food regulation?

India is silent on the fat tax debate. It would like to stay fat and unfit.

In the High Court of Delhi, responding to the demand to ban junk food in schools, the government, through the FSSAI, took cover behind technicalities.

According to it, junk food is not defined, but instead falls within the category of proprietary food – which is food not standardised under existing regulations. This category of food is only expected to declare its composition or nature and comply with general regulations under the Food Act.

The government has also taken the 'weak' plea that Food Safety and Standards Act 2006 does not "empower the authorities to ban any particular category of food". It also takes the usual position of pushing responsibility of any action on to the state government: this, after it enacts a law which centralises responsibility! The affidavit filed by government says: "It is the responsibility of the state food safety authorities to monitor and verify that the relevant requirements of law are complied with." In addition, it is the school which should ban junk food.

In other words, the government can and will not do anything to protect public health.

20. What is the status of the court case against junk food in schools? What are the regulations on junk food in India?

In December 2010, the Uday Foundation for Congenital Defects and Rare Blood Groups, a non-profit based in New Delhi, filed a Public interest Litigation (PIL) in the Delhi High Court. The PIL demanded an immediate ban on junk food and carbonated beverages in canteens of schools and colleges, and a ban on the sales of these junk food around 500 yards of the school; the development of a canteen policy for schools and ban on advertisements of junk food and carbonated beverages in television and all other media.

The PIL triggered a debate on junk food in the country. On November 2, 2011, the Union of India filed an affidavit accepting "in principle that there is a need to ban junk food in schools". According to the affidavit filed by the Food Safety and Standards Authority of India (FSSAI) in the High Court, "junk food has not been defined under the Food Safety and Standards Act, 2006. So the food items like burgers, pizzas, *samosas*, fries and the rest fall under the category of proprietary food. The Food Safety and Standards Act 2006 defines 'proprietary and novel food means an article of food for which the standards have not been specified but is not unsafe'."

The affidavit laid out the steps which have already been taken to achieve this, including writing to states and government schools to ban junk food.

Listening to this, the High Court directed: "We are not satisfied with the so-called efforts made by the Union of India in this direction. We would like the Union of India to take concrete and effective steps to ensure that the sale and supply of junk food in and

HEALTH MINISTER GHULAM NABI AZAD WRITES TO EDUCATION MINISTER KAPIL SIBAL, DECEMBER 2011

I am writing to draw your kind attention to our letter of even number dated 31.1.2007 (copy enclosed) indicating the increasing trend, particularly among school and college students and adolescent population, to consume junk food and carbonated drinks, which are rich in calories but deficient in vital nutrients. This has become a major public health concern world-wide, as these foods and drinks provide only energy but little or no vitamins and minerals.

4. In this context, I would request you to kindly seriously consider issuing appropriate directions to the schools and colleges for promoting healthy food habits and discouraging foods high in saturated fat and carbonated drinks from their canteens. Children must be made aware of the benefits of consumption of fruits, vegetables and whole grains.

around schools is banned."

When the court met again on January 11, 2012 the FSSAI on behalf of the Union of India mentioned that it is undertaking a project for development of guidelines for making available quality and safe foods in schools. These guidelines have to be submitted in court in July 2012.

21. What are schools doing to ban junk food?

In December 2011, the Union health minister, Ghulam Nabi Azad wrote to his colleague, Union human resource minister, Kapil Sibal reminding him of a letter written way back in 2007 about the growing use of junk food and carbonated drinks, "rich in calories but deficient in vital nutrients", in schools. This, he said, has become a "major health concern as these foods and drinks provide energy but little or no vitamins and minerals".

He then went on to request the education minister to issue "directions to schools and colleges to promote good food and discourage foods high in saturated fat and carbonated drinks in their canteens."

However, there is no evidence that any action has been taken on the basis of this letter.

What is encouraging, however, is that many schools, particularly in Delhi, have taken steps to ban junk food and carbonated beverages in their premises. Clearly, this will be the way ahead – particularly as the government seems to be disinterested in protecting people's health.

But it is time that governments took decisive steps. There is a clear consensus that there is a need to replace junk food with nutritious food in schools and outside. It is important that there be policies on advertising and rules that ensure that advertisement of junk food is restricted or prohibited during children's programmes and on cartoon channels. 'Bad' food should be taxed and the money should be used to advertise nutritious and healthy foods.

22. What are the new dangers of obesity and environment and why should we be particularly worried?

There is increasing evidence that 'fat' people have another health burden to worry about. Researchers are now finding convincing evidence of "chemical obesogens" – industrial chemicals, pesticides, air pollutants – that in turn alter metabolic processes and predispose people to gain weight. In other words, chemical exposure in the environment could be contributing to the obesity epidemic. But this cycle is even more deadly, as 'obese' people are more vulnerable to toxins.

The February 2012 issue of *Environmental Health Perspectives* discusses the growing danger of obesogens.[38] The main role of fat cells is to store energy and release it when needed. Scientists also know that fat tissue acts as an endocrine organ – releasing hormones related to appetite and metabolism. Research suggests that different obesogenic compounds have different mechanisms of action, some affect the number of fat cells, others affect appetite and some pass on changes to later

generations. Early-life exposure to certain obesogens could alter individual metabolism and fat cell make up for life.

But the story does not end here: obesogens make getting thinner even more difficult. The April 2012 edition of *Physiology and Behavior* finds that fat loss triggers release of obesogens back into the bloodstream, at which point they exert influence in a number of ways. They can suppress thyroid function – a key factor in our body's energy burning capacity. They can also lead to a decline in the body's oxidative capacity – the ability to burn fat in muscle. Ultimately, our resting metabolic rate, the number of calories that our bodies burn without any movement or exercise, drops.[39]

There is now a growing list of potential obesogens – from pesticides like DDT to fine particulate matter, which is emitted from diesel and other sources. A study on bisphenol A (BPA), used in the plastic industry, found that this chemical reduces fat cells, but programmes them to incorporate more fat so there are fewer but very large fat cells. This then increases abdominal fat and glucose intolerance. Given Indians' propensity for central obesity, clearly, growing pollution in our food and air is the trigger we do not need.●

4.2 HOW JUNK IS OUR JUNK FOOD

Junk food consumption in India is growing unchecked at 35-40 per cent annually. Junk food falls under the category of proprietary foods, the government doesn't regulate them and there is no one to check the veracity of claims they make on their labels. CSE's Pollution Monitoring Lab decided to do an independent report on the nutritional constituents of the junk food commonly sold in the market – from potato chips and fries to burgers, pizzas and soft drinks. The results show why junking junk food should be the most important public health agenda of the country.

1. What is the CSE study on junk food?

Junk food refers to food which is easy to make and quick to consume. These foods have no nutritional value and are often high in fat, salt, sugar, and/or calories. Common junk foods include salted snack foods, fried fast food, and carbonated drinks. Most junk food have trans fats; these trans fats behave like saturated fats when they get in the body, clog up the arteries and cause plaque to build up, contributing to heart diseases and strokes.

Junk food is unhealthy because it is high in calories, fat and trans fats, sugar in liquid form and salt, and low in fibre and nutrients. CSE's study was aimed at establishing how much of the unhealthy constituents these junk foods have and how correct are their nutritional claims.

CSE's Pollution Monitoring Lab (PML) collected 22 samples of most popular brands of packaged junk food available in the market – three of potato chips, two of Indian snacks, two of instant noodles, two of carbonated beverages, six of burgers, three of pizzas, three of potato fries and one of fried chicken – and tested them for total fats, trans fats, carbohydrates, salt and sugar.

2. What methodology was used by PML to test?

The PML used internationally accepted methodologies to test fats, trans fats, salt and carbohydrates.

- **TOTAL FAT:** The Association of Official Analytical Chemists International (AOAC) method (922.06) was used for the total fat analysis.
- **TRANS FATS:** The Association of Official Analytical Chemists International (AOAC) method (996.06) and gas chromatography was used for the trans fat analysis.

- ***SALT:*** The Association of Official Analytical Chemists International (AOAC) method (937.09) was used for determination of salt (chlorine as sodium chloride).
- ***TOTAL CARBOHYDRATE:*** Total carbohydrates in food were estimated by the anthrone method, which is a colorimetric method.

3. *What were the findings of the CSE study?*

The results of the study indicate that junk food contains high levels of sugars, salt, total fats and trans fats.

- Highest level of total fat was found in an Indian snack (Haldiram's Aaloo Bhujia): 37.8 gram (g) per 100 g of the sample.
- Trans fat content was the highest in French fries (8.1 per cent of the total fat), followed by instant noodles (4.6 per cent of the total fat) and potato chips (4.5 per cent of the total fat).
- Salt content was the highest in instant noodles (3.7 g per 100 g of sample). Consumption of a packet of instant noodles, therefore, will cover about half of the daily salt quota. The salt content was not declared by the companies on the label.
- The highest level of carbohydrates was detected in Top Ramen noodles at 73.3 g per 100 g.

4. *Are companies making misleading claims on their product labels and flouting the law?*

India does not define junk food properly. According to the Food Safety and Standards Act of 2006, foods like burgers, pizzas and chips fall under the category of "proprietary or novel" foods, for which standards have not been specified; but these are not considered unsafe. As a result, makers of this category of foods declare their composition only broadly. Fast food giants take advantage of this provision to dupe consumers. Even though the Food Safety and Standards Authority of India (FSSAI) mandates food companies to declare information on total energy, carbohydrates and sugars, proteins, fats and trans fats on the product label, the study by CSE reveals that consumers are being led on to believe what the companies want them to believe.

Flouting norms: Haldiram Aloo Bhujia and Top Ramen instant noodles claim to be trans fats-free. But the CSE study found they are not. As per FSSAI rules, a product can claim to be trans fats-free if it contains less than 0.2 g of trans fats per serving. The CSE study found that a packet of Top Ramen instant noodles has 0.6 g of trans fats; 100 gm of Haldiram Aloo Bhujia has 2.5 g of trans fats. Haldiram claims its serving size is 10 g, but it is nothing but a gimmick. Consumption habits of people exceed this serving size and they end up consuming more trans fats.

Misleading claims: Many brands put misleading information about their trans fats content on the product labels. Products like Lay's American Style Cream & Onion claim that they have "zero" trans fats in 100 g of their products. However, the CSE study found 0.9 g trans fats in 100 g of the product. Similarly, brands like Haldiram Aloo Bhujia, Bingo Oye Pudina chips and Top Ramen noodles claim they have "zero trans fats" per 100 g of product. But the CSE results show otherwise.

No standardisation: Lay's American Style Cream & Onion chips carries nutritional information for two serving sizes (14 g and 100 g) on its label. But Uncle Chips Spicy Treat does not mention the nutritional information for a serving size. It just provides the data for 100 g. Lack of nutritional information for a serving size leaves one with no scope for knowing how much calories he or she consumes.

AVERAGE NUTRITIONAL VALUES IN JUNK FOOD

Sample name	Total carbohydrate (g/100 or %)	Salt (g/100g or %)	Total fat (g/100 or %)	Trans fat (g/100 g of food)
Potato chips	57.5	2.3	32.8	0.8
Indian snacks	49.9	2.5	35.9	1.6
Instant noodles	71.6	3.7	14.1	0.6
Carbonated drinks	14.4			
Burgers	38.2	1.5	11.9	0.4
Pizza	50.3	1.0	7.1	0.1
Fries	56.5	0.4	19.9	1.6
Fried chicken	14.0	0.9	23.4	0.7

Source: CSE Lab study

Fat of the matter

FRIED POTATO CHIPS

It has around 33% fats. This means if one munches a standard-sized packet of chips (65-75gm), he or she consumes about half of the daily fats quota. Unlike in a balanced diet, where a maximum of 30% of calories should come from fats, 50-60% of calories come from fats in chips

INDIAN SNACKS

Fats and carbohydrates combined, 100 gm of Kurkure has enough calories to satisfy one-fourth of one's daily recommended quota. If you are fond of *aloo bhujia* with tea, you get high doses of salt and trans fats, along with a high amount of calories

INSTANT NOODLES

The "tasty and healthy" meal comes with high salt, empty calories. A packet of noodle has around 3 gm of salt; recommended intake is 6 gm/day. Addition of vitamins, as claimed by Maggie Noodles, doesn't make it a healthy food as it has negligible fibres; 70% of it is just carbohydrates

POTATO FRIES

Fries that one eats with burger and soft drink are laden with fats: 20% of its weight is fats, 1.6% of its weight is trans fats. By eating a large serving (220 gm), one exceeds the safe limit for trans fats. Additional trans fats come from accompanying burger too

Nutritional guidelines

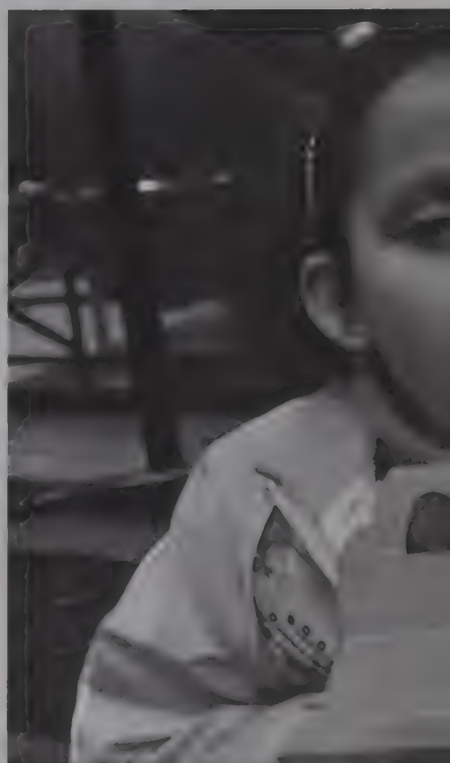
Person	Kilo calories allowed/day	Carbohydrate (gm/day)	Salt (gm/day)	Total fats (gm/day)	Trans fats (gm/day)
Adult male	2,320	290-348	6.0	39-78	2.6
Adult female	1,900	263-315	6.0	35-70	2.1
Children (10-12 yr)	2,100	238-285	6.0	32-64	2.3

CSE's findings

Sample	Total carbohydrate (gm/100gm)	Salt (gm/100 gm)	Total fats (gm/100 gm)	Trans fats (gm/100 gm)
POTATO CHIPS				
Uncle Chipps Spicy Treat	52.2	3.5	34.3	0.8
Lay's American Style Cream & Onion	56.9	1.2	33.1	0.9
Bingo, Oye Pudina	63.4	2.3	31.0	0.6
INDIAN SNACKS				
Haldiram's Aloo Bhujia	45.6	3.3	37.8	2.5
Kurkure Masala Munch	54.2	1.6	34.0	0.7
INSTANT NOODLES				
Masala Maggi	69.8	4.2	14.0	0.6
Top Ramen Super Noodles (Masala)	73.3	3.2	14.3	0.7
CARBONATED DRINKS				
Pepsi	14.0	0.0	0.0	0.0
Coca Cola	14.8	0.0	0.0	0.0
BURGERS				
McAloo Tikki (with cheese)	48.1	2.0	8.3	0.3
KFC's Veg Zinger (with cheese)	45.4	1.7	13.7	0.7
Nirula's Subz Burger (with cheese)	36.8	1.7	9.6	0.3
McChicken	37.6	1.1	10.7	0.4
KFC's Chicken Zinger	30.2	1.2	16.9	0.5
Nirula's Chicken Burger	31.0	1.2	12.2	0.3
PIZZA				
Pizza Hut's Margherita Pan	51.6	1.4	7.9	0.1
Domino's Margherita	43.7	0.6	6.3	0.1
Slice of Italy's Margherita Classic	55.6	1.0	7.0	0.1
FRIES				
McDonald's Fries	55.8	0.3	16.6	1.3
KFC's Fries	59.0	0.8	19.0	1.7
Nirula's French Fries	54.8	0.2	24.1	1.8
FRIED CHICKEN				
KFC's Hot and Crispy	14.0	0.9	23.4	0.7

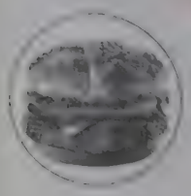
Fatal combos

Companies serve most of the ju food in combinations. For childr they have "happy meals". To m it more alluring, they throw in a extra toy. After all, it's all about the image of a happy family. Bu what is it doing to your insides? Take a look



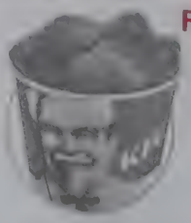
PHOTOGRAPHS: RAVI BATRA

Methodology: Internationally acceptable methods were used to estimate the level of fats, trans fats, salt and carbohydrate in the food. Total fats, trans fats and salt were estimated using method numbers 922.06 996.06 and 937.09 respectively as provided by the Association of Official Analytical Chemists. Total carbohydrates were measured using the Anthrone method



BURGER

KFC's Chicken Zinger has 16.9% fats. McAloo has 8.3% fats. How unbalanced diets are they is gauged from the fact that 35% of calories in a veg burger come from fats. In non-veg burgers 47% calories are from fats



FRIED CHICKEN

Regular consumption of this product is likely to make one obese. A two-piece fried chicken of KFC (about 250gm) has nearly 60 gm of fats, which is recommended for the whole day

CARBONATED DRINKS

The 300 ml serving that one drinks with all kinds of junk food has enough sugar (over 40 gm) to exceed one's daily sugar quota of 20 gm. After this, forget the cup of tea, one should not even eat fruits. Any additional sugar will make one fat

PIZZA

By far, pizzas were found to be healthy compared to the other junk foods tested. They have low levels of salt and fats; levels of trans fats were also low. CSE tested only the basic pizza. Those with extra cheese are more popular and might not be very safe

- Carbs
- Sugar
- Fats
- Trans fats
- Salt
- % of daily kilo calories need met

KFC MEAL

Contents: 1 Chicken Zinger burger + 1 piece of chicken (around 100 gm) + fries (100 gm) + carbonated drink (300 ml)

Carbs	Sugar	Fats	Trans fats	Salt	% of daily kilo calories need met
156.6 gm	43.2 gm	63.9 gm	2.9 gm	3.3 gm	57%

This meal will stuff you with fats and trans fats, and take care of 60-70% of your calorie requirement for the day

MCDONALD'S MEAL

Contents: 1 McChicken (157 gm) + Fries (110 gm) + carbonated drink (300 ml)

Carbs	Sugar	Fats	Trans fats	Salt	% of daily kilo calories need met
163.1 gm	43.2 gm	35.1 gm	2 gm	2 gm	47%

This meal will meet more than 50% of a child's calorie requirement for the day

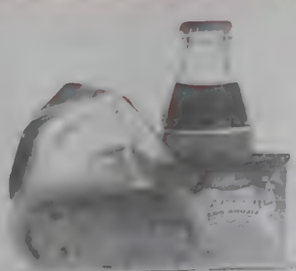
PIZZA MEAL

Contents: 1 pizza (250 gm) + carbonated drink (300 ml)

Carbs	Sugar	Fats	Trans fats	Salt	% of daily kilo calories need met
168.4 gm	43.2 gm	17.5 gm	0.2 gm	2.4 gm	41 %

This meal might be fine as long as you do not order extra cheese or add cheesy side-dishes

COLLEGE MEAL



Contents: Instant noodle (1 packet) + *aloo bhujia* (50 gm) + carbonated drink (300 ml)

Carbs	Sugar	Fats	Trans fats	Salt	% of daily kilo calories need met
123.2 gm	43.2 gm	30.2 gm	1.7 gm	4.6 gm	38%

The salt level in this meal is so high that it meets 75% of the daily recommended value

SMART SNACKS: NUTRITION JUGGLERY IN CLAIMS

Till February 2012, Frito Lay's products claimed to be "smart snacks" because they used a healthy oil. At that time, the labels clearly mentioned that the products were trans fats-free. Which was incorrect though. When CSE tested Lay's American Style Cream & Onion chips, it found that 100 g of the chips contained 0.9 g of trans fats.

But from March onwards, the product ceased to be trans fats-free. This was done very surreptitiously: the pack remained the same, Saif Ali Khan continued to call it 'Dil logical' but the 'smart snack' tag went missing. There were no more claims of being zero trans fat. This skipped the advertisement blitz. When CSE tested the sample of this new Lays pack, the trans fats were very high: 3.7 g/100 g.

No information: Non-packaged junk foods, such as burgers, pizzas etc, give nutritional information on their websites, but there seems to be a double standard in play. McDonalds, for instance, gives information on 22 nutritional attributes on its American website. This includes information on different types of fats and the levels of trans fats in the product. On the other hand, the McDonald's website for India provides information only on six nutritional attributes. Most importantly, it fails to provide any information on trans fats. But the company at least provides information on portion sizes in India. Other companies do not even do that. Pizza Hut's website for India is a case in point. The company's website in the US provides 12 nutritional attributes of the product, including the serving size and trans fats. Its website for India provides only six attributes in the dining menu; it does not mention trans fats. Its delivery menu does not provide any nutritional information. KFC's website in US also provides the information on 12 nutritional attributes including the serving size, types of fats, including trans fats, and fibres. But its Indian website gives only four nutritional information.

Serving size: While a few give details on the serving size, they are bordering on the ridiculous. For instance, Lays chips recommends that the serving size should be 14 g which is about 10 chips; Haldiram's Aloo Bhujiya recommends that the serving size should be 10 g, which is just about a fistful. The serving sizes vary from country to country: while Lays in the US recommends a serving size of 28 g, the Indian serving size is fixed at 14 g.

5. *What should India do to regulate and control the health impacts of junk food?*

Many countries have imposed restrictions on the sale of junk food in and around schools and put in place 'fat' taxes to dissuade consumers from consuming junk food. India too should take some lessons.

- Ban junk foods from schools and places where children can have easy access to them.
- Ban junk food advertisements in children's programmes; implement stricter advertisement guidelines for children's TV channels, and allow on other channels only after 9 pm.
- Impose a 'fat tax' on food items like processed food if they contain more than 2.3 per cent saturated fat.
- The FSSAI should come out with regulations to reduce salt, sugar and fats in junk foods, including imposition of 'fat, salt and sugar tax'.
- Change labeling rules for trans fats and salt.

4.3 THE FAT OF THE MATTER

Which cooking oil is best for us? We are bombarded with advertising messages telling us a particular healthy oil is good for our heart. These advertisements talk of monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) and of course, catch-us-words like omega properties. Food is, after all, nutrition and even medicine. It must be taken seriously. So, we as aware citizens go out and buy the healthy oil.

But the truth is that we only know what companies selling their products are telling us. Centre for Science and Environment's Pollution Monitoring Laboratory tested various types—from peanut and mustard, to safflower, sunflower, olive and more. The objective was to not only understand the fat of the matter, but also to check for quantity of trans fatty acids, a particularly noxious ingredient in the oil we eat. As the results came in, we compared them with what we knew about these oils. It is then we realised we do not even begin to understand the science of our food and its relation with our bodies—in a world, where our food is not our own anymore. The business is in our kitchen. In this business, our nutrition and its science are also a business, even profit. If food regulators slip — are unmindful or negligent — our health is compromised.

The story of our cooking oil is about our bodies. CSE researchers started with the presumption, verified by nutrition regulators, that a healthy oil is one that has less saturated fat, more MUFA and the level of PUFA is balanced between saturated and mono. In addition, we need to consider the sub-constituents, the essential fatty acids — omega 6, omega 3 and the current posterboy, omega 9. The oil which has these in some proportions, is the best. We thought we had cracked it. But no, there is much more to the art and science of oil in our food. Read to learn what we found and what governments and you need to do about it.

1. What is healthy oil?

The oil we eat is essential for our bodies to function. We cannot do without it. So, it is important to understand its chemistry. It is agreed that healthy oil is one which has less saturated fat, more monounsaturated fat (MUFA) and polyunsaturated fats (PUFA) which is balanced between the two. It should also be rich in omega 3, 6 and 9 fatty acids (see Box: *The bare essentials of the oils we ingest*).

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THE BARE ESSENTIALS OF THE OILS WE INGEST

Fats and oils are made up of chains of carbon atoms. The bonds between the carbon atoms classify the oil into saturated and unsaturated fats.

Saturated fats have a single bond between carbon atoms (they have more hydrogen atoms).

Unsaturated fats have double bonds between the carbon atoms. Where double bonds are formed, hydrogen atoms are eliminated and energy is released.

The unsaturated fats are further classified into monounsaturated fats (MUFA) containing one double bond and polyunsaturated fats (PUFA) containing more than one double bond.

MUFA includes omega 9.

PUFA includes omega 6 and omega 3.

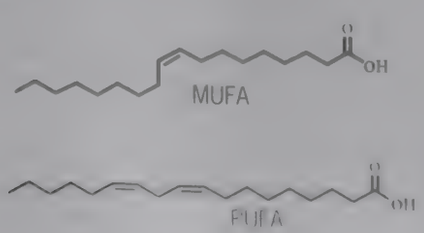
The body needs these essential fatty acids to manufacture and repair cell membranes, to enable the cells to obtain optimum nutrition and expel harmful waste products. It is now accepted that omega 9 found in MUFA, could be the best.

In relation to our health, it is broadly accepted that PUFA protects against cardiovascular diseases by providing more membrane fluidity than MUFA.

On the other hand, it is also found that foods with MUFA lower LDL (bad) cholesterol, while possibly raising the HDL (good) cholesterol.

Trans fats are produced when unsaturated fats are partially hydrogenated. These hydrogen atoms saturate the double bonds and also change the configuration to trans.

While both MUFA and PUFA oils can give rise to trans fats, oils rich in PUFA have more double bonds and result in fats rich in trans fats.



2. *Which is the best oil?*

It is impossible to say with certainty what oil is the best. The reason is partly to do with commercial interests, which give oil's good and bad labels. This is because big interests are involved in growing and selling a particular oil. So, the politics of oil in our kitchen must be understood before deciding on what to buy.

3. *What is the global politics of oil?*

Certain good oils have been given a bad name, simply because there is no one to 'fight' for them. For instance, coconut oil, which has a high amount of saturated fat, scores poorly on this good-oil matrix. But studies found that the oil is healthy because its carbon chains are shorter in length than other saturated fats. A study in Kerala in 1995 on 64 volunteers showed that adding coconut kernel to the diet did not bring about any change in the total cholesterol, HDL cholesterol or LDL cholesterol, in the serum.

Research has also found that coconut oil has antimicrobial components and leads to normalisation of body lipids, protects against alcohol damage to the liver and improves the immune system's anti-inflammatory response.

But it is still not the preferred medium for the market because coconut oil is expensive; it is shunned by the food industry. The industry prefers oils like canola (rapeseed), says Mary Enig, a nutritionist in the US, who writes on fats and their impacts on human bodies. She says this industry-friendly oil was marketed as being "as healthy as olive oil" because it is similarly rich in MUFA. But then research found that about two-thirds of the MUFA in rapeseed is erucic acid, which was associated with fibrotic lesions in the heart. To fix this, Canadian plant breeders developed a variety of rapeseed low on erucic acid and high on omega 3 and oleic acid, which is an omega 9 fatty acid.

This new oil, sold as lear oil, bombed in the US as farmers and consumers shunned it. Industry then went to town to sell and decided to change the name — from 'rape' and 'lear' to canola, to give a healthy image for what Enig calls the Cinderella oil.

All of a sudden, canola oil began to appear in recipe books as something which was perfect for Mediterranean diets, replacing olive. Research and recipes were pushed, this time extolling omega 3; canola is high on this fatty acid.

Enig notes that this hard-sell was not right. A large numbers of studies point to the fact that "canola oil is definitely not healthy for the cardiovascular system". She points out that these studies also find that the use of this oil retards

growth, which is why the US Food and Drug Administration does not allow it in infant formula.

But then, how were people in China, Japan and India, where rapeseed oil has been traditionally used, coping? Research has found that the adverse impacts of rapeseed oil can be mitigated if it is combined with butter or ghee. The problem is not with the content of the erucic acid, but with the high levels of omega 3 in the absence of saturated fats. In other words, explains Enig, the real problem was the advice given to Indian housewives not to use butter and ghee, which were used traditionally in combination with rapeseed oil.

Another problem is the way the oil is processed. In the past, rapeseed oil was cold pressed in neighbourhood mills and consumed fresh. This, Enig says, gave it its added benefits.

4. Is cold pressed oil better than processed and refined oil?

This is another debate in the science of oil and nutrition. Olive oil is considered the best for the heart only when it is cold pressed. Research published in the open-source journal BMC Cancer, done by Spanish researchers, found that the major complex phenols present in extra-virgin olive oil also prevent cancer. These are not present when the oil is extracted through solvents.

In India, there is a similar perception about cold-pressed mustard oil, which is considered better. While health reasons are propelling a growing interest in other traditional oils like rice bran and coconut, these are shunned by the modern oil industry.

5. What is the importance of omega factors in oil?

While there is a growing attention on the omega factors in oil, researchers cannot agree on what works best in which circumstance. It is accepted that the human body needs a ratio of 5-10:1 of omega 6 to omega 3. A P Simopoulos from the Center for Genetics, Nutrition and Health, Washington DC, in the September 1999 issue of *American Journal of Clinical Nutrition*, found that over the past few years, there has been an enormous increase in the consumption of omega 6 fatty acids due to the increased intake of vegetable oils from corn, sunflower seeds, safflower seeds, cottonseeds and soybeans. As a result, today's Western diet has a ratio of omega 6 to omega 3 of 30:1.

But the jury is still out on what is the best ratio of omega 6 to omega 3. It is clearly the issue to understand better in the years to come.

6. Then what oil should we buy?

Frankly, there is no straight answer for this question. In spite of all the hard-sell for one type of oil being the best, nutritionists are of the view that the best oil is the oil used in moderation and switched frequently to get the maximum nutrition value.

D Prabhakaran, executive director of the Centre for Chronic Disease Control in Delhi suggests combining mustard, canola and peanut oil. Swati Bhardwaj, nutritionist and diet consultant with the Diabetes Foundation (India), says canola, olive and rice bran oils are the best options available in the market for Indians. Others say the best option would be to use oils rich in MUFA, like olive, mustard and groundnut as well as those rich in PUFA, like soyabean and sunflower.

But the important issue is that the choice must be yours (and not the company's) to make (see Box: *Your guidebook to oils*).

7. What are South Asians eating?

Anoop Misra, director, department of diabetes and metabolic diseases, Fortis Hospital, New Delhi, and his colleagues have reviewed the oils and fats that South Asians are consuming and the linkages with human health.

They found several studies had reported diets in India which had higher intakes of carbohydrates, saturated fatty acids and omega 6. These studies also found low intakes of MUFA, omega 3 and fibers. Intervention studies, which added omega 3 to the diet, improved the lipid profile, but did not show a beneficial effect on insulin resistance.

The problem, Misra's study points out, is that South Asians, particularly Indians, are prone to developing insulin resistance. Therefore, the current diet is contributing to the development of metabolic syndrome and diabetes. The doctors advise it is critical for Indians to replace unhealthy fast food with healthy options.

8. Who regulates oil for health and safety?

The question is, how is the oil we consume regulated in the country. There are a number of players — from the Food Safety and Standards Authority, which regulates the Food Safety and Standards Act to the Bureau of Indian Standards, which has a voluntary standard for oil, the Agmark (the Agricultural Produce Grading and Marking Act) that set guidelines for its sale. In September 2008, the government revised its labelling requirements for food, which include provisions for companies to publish nutritional information. Under this notification, companies have to publish information on the different fatty acids, including trans fats, MUFA and PUFA, in their product.

The regulation leaves a lot of room for

YOUR GUIDEBOOK TO OILS

On the basis of results on fatty acid profile of different oils in the market, CSE presented a matrix which you could use to figure out what to cook your food with. The matrix ranks the oils on the basis of saturated fats – the lesser the better – and unsaturated fats like MUFA, PUFA and essential fatty acids like omega 3, 6 and 9 – the more the better. The laboratory study also provided classification of the oils on the basis of WHO's recommendations. These are based on the ratio of PUFA and saturated fats – the ratio should be between 0.8 and 1. Another ranking is on the basis of ratio of omega 6 and omega 3 – this should be between 5 and 10.

Saturated fatty acids		Monounsaturated fatty acids		Polyunsaturated fatty acids		Omega 9		Omega 6		Omega 3		PUFA/SFA		Omega 6/ Omega 3	
Mustard	4.45	Olive	66.97	Soybean	53.21	Olive	65.56	Safflower	49.37	Soybean	10.89	Mustard	6.82	Blend	117.3
Safflower	8.53	Mustard	49.99	Safflower	50.09	Groundnut	40.60	Soybean	39.07	Mustard	7.09	Safflower	5.88	Sesame	88.49
Sunflower	13.46	Groundnut	43.08	Sesame	39.18	Sesame	38.99	Sesame	38.05	Ricebran	6.20	Soybean	3.43	Safflower	88.20
Olive	14.15	Sesame	38.99	Blend	34.57	Ricebran	36.43	Sunflower	33.06	Groundnut	3.31	Sunflower	2.50	Sunflower	65.66
Soybean	15.51	Ricebran	36.58	Sunflower	33.63	Sunflower	35.23	Blend	30.50	Butter	2.43	Sesame	2.34	Palm	23.89
Blend	15.78	Sunflower	35.32	Ricebran	31.37	Palm	33.70	Groundnut	24.74	Desi ghee	1.03	Blend	2.20	Vanaspati	13.27
Groundnut	16.09	Palm	33.70	Mustard	30.33	Blend	24.91	Ricebran	24.69	Safflower	0.56	Groundnut	1.78	Groundnut	7.54
Sesame	16.77	Blend	25.33	Groundnut	28.60	Vanaspati	18.30	Mustard	9.98	Sunflower	0.50	Rice bran	1.24	Desi ghee	6.71
Rice bran	25.38	Desi ghee	21.10	Desi ghee	10.63	Soybean	18.11	Palm	9.92	Sesame	0.43	Olive	0.56	Rice bran	3.98
Vanaspati	41.26	Butter	18.96	Palm	10.52	Desi ghee	17.77	Olive	6.91	Palm	0.42	Palm	0.23	Soybean	3.59
Palm	45.82	Vanaspati	18.66	Vanaspati	8.18	Butter	15.23	Desi ghee	6.91	Blend	0.26	Vanaspati	0.20	Mustard	1.41
Desi ghee	55.94	Soybean	18.18	Olive	7.94	Safflower	11.44	Vanaspati	2.37	Vanaspati	0.18	Desi ghee	0.19	Butter	0.52
Butter	59.44	Safflower	11.44	Butter	4.07	Mustard	7.62	Butter	1.26	Olive	0.00	Butter	0.07	Olive	0.00
Coconut	88.89	Coconut	6.74	Coconut	1.04	Coconut	6.74	Coconut	1.04	Coconut	0.00	Coconut	0.01	Coconut	0.00

Note: Blend — Blended Safflower + Rice bran oil; Average value of different oils tested by CSE lab

manoeuvre. As a result, companies can give the composition in a range — Rath Vanaspati says its package has saturated fatty acids in the range of 16-54 per cent; MUFA 36-65 per cent; PUFA 3-44 per cent and trans fats 8-33 per cent. Others print values that are standard compositions of oils found in academic literature. The CSE laboratory study detected differences between the labelling claims and its analytical study. Who checks the companies' claims? Do we have MUFA-PUFA inspectors?

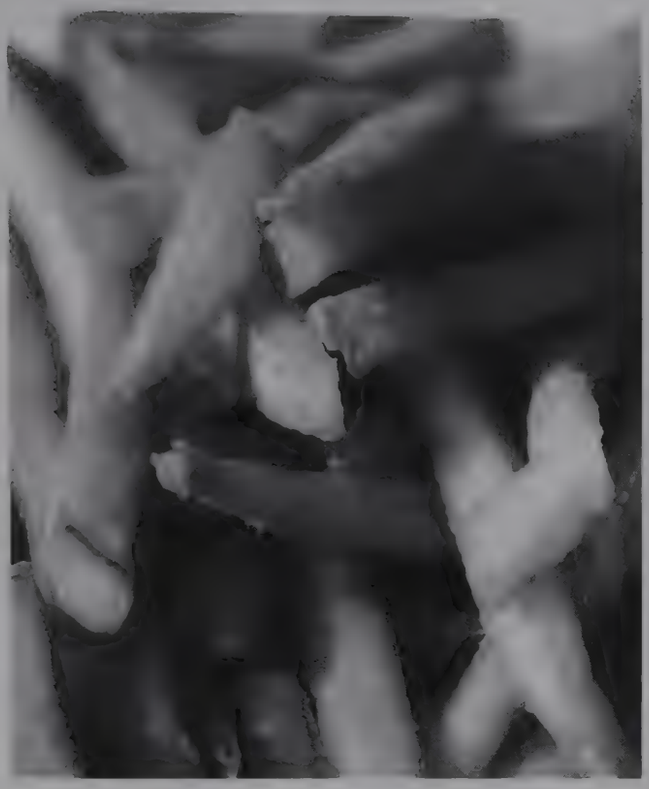
The Directorate of Vanaspati, Vegetable Oils and Fats has supposedly got inspectors to check quality. In addition, the food and drug administrations of different state governments are required to inspect food that is sold to enforce the provision of Food Safety and Standards Act. There are also inspectors of the directorates of marketing and inspection to check grade, quality and packaging. Seems there is no shortage of inspectors. But are they checking?

As there are no mandatory standards for oil manufacturers, it is anybody's guess what is checked and what is bottled. In mid-2008, the UK Food Standards Agency claimed it had found significant amounts of mineral oil mixed in the sunflower oil being sold in the country. It asked for this brand to be recalled. But India does not do such checks. The rule is what we do not know, does not hurt. But it does hurt, we just don't know.

WHO MAKES THE OIL WE EAT?

- RATH: **Agro Tech Foods Ltd, Secunderabad, affiliated to ConAgra Foods Inc of USA, one of the world's largest food companies. It acquired Rath vanaspati brand from Siel Ltd.**
- DALDA: **Now made by Bunge Limited, a mega-food giant in the US. World leader in agribusiness, fertilizer and food products, the multinational bought Dalda from Hindustan Lever.**
- GEMINI: **By Cargill Inc in the US, which deal in seeds and genetic food, among other things. Largest selling edible oil brand in Maharashtra, according to A C Nielsen Retail Audit November 2007.**
- RAAG: **Joint venture between the Adani group in Gujarat and the Wilmar International Limited in Singapore. The Adani-Wilmar owned Fortune brand was India's No 1 edible oil brand in 2006, said the A C Nielsen Retail Audit November 2006.**
- JINDAL: **Jindal group, with a wide manufacturing range, from steel to electric goods.**
- GAGAN: **Amrit Banaspati Company, Chandigarh; claims to be India's largest selling vanaspati.**
- PANGHAT: **Part of the Siel group, Mawana Sugars Ltd is its new name.**

4.4 THE TRUTH ABOUT TRANS FATS



Oil is essential for our body to function. But when CSE tested the cooking mediums used in our food, it discovered that the branded edible oils were full of unhealthy trans fats. Trans fats are formed during the process of addition of hydrogen atoms to oils, a process industry prefers as it keeps the oil from turning rancid and ensures a longer shelf life. CSE's study results showed that trans fats in seven leading vanaspati brands were five to 12 times the 2 per cent standard set by Denmark. The standards set by Denmark are the most stringent.

Trans fats are associated with a host of serious health problems ranging from diabetes and heart diseases to cancer; and this is reason enough to regulate them. The Indian government has been 'considering' the issue of trans fatty acids in vanaspati for nearly eight years. Under pressure, the health ministry came out with a notification in 2008 for labeling food including trans fats. But without any standards, this notification remains toothless.

1. *What are trans fats?*

Trans fats or trans fatty acids (TFA) are formed during the process of adding hydrogen atoms to cooking oil.

In the 1970s, when concern about the link between saturated fats (like butter, lard, tallow) and heart diseases started growing, experts suggested a shift to unsaturated fats, which are in liquid form. But the food processing industry needed semi-solids for products like cakes and biscuits, for which they partially hydrogenated the oil. Hydrogenation is the addition of hydrogen atoms to break oil's double bond. In oils, the hydrogen atoms are on the same side of the double bond of the carbon chain – 'cis' (from Latin: on the same side). During partial hydrogenation, the chain is twisted in a way that the hydrogen atoms end up on different sides of the chain – 'trans' (from Latin: across). The product is a semi-solid which carries the unhealthy component, trans fats. Industry needed hydrogenation because it ensures that its product is less likely to turn rancid, can withstand repeated heating and food cooked in it stays fresh.

2. *In 2008, the CSE lab conducted a study of trans fats in cooking oils. What had prompted the lab to do this study?*

CSE's study was driven by a concern for public health. A large proportion of India's population is

WHAT TRANS FATS DO TO US

Trans fats are bad for health. They are especially bad for the heart, as they reduce the amount of good cholesterol (HDL). They can also increase the risk of breast cancer and infertility in women along with diabetes and Alzheimer's disease.

- **Heart diseases:** Metabolic studies have shown that trans fats have adverse impact on blood lipid levels, increasing the LDL (or bad) cholesterol while decreasing the HDL (or good) cholesterol. The strongest epidemiological evidence relating intake of trans fatty acids to the risk of coronary heart diseases (CHD) has been reported from three large cohort studies, the Health Professionals Follow-up Study (HPFS), the Alpha-Tocopherol Beta-Carotene study (ATBC) and the Nurses Health Study (NHS).
- **Cancer:** In the EURAMIC study dating from 1997, the association between trans fatty acid levels in adipose tissue and the incidence of cancer was investigated in European populations. The study found a link between intake of trans fats and cancers of the breast and large intestine.
- **Diabetes:** Clinical studies show that trans fatty acids can increase insulin resistance. There are also experimental data showing that most, but not all, animals consuming a high fat diet will become obese.
- **Effects on fetus:** Recent studies on humans have shown that trans fatty acids are transferred to the fetus, as they have been found in the same levels in the blood of newborn infants as in that of mothers. Both the fetus and the breast-fed baby are consequently exposed to trans fatty acids corresponding to the mother's intake. Dietary trans fatty acids can in part compete with essential polyunsaturated fatty acids in the body.

  In animal experiments, a high intake of industrially produced trans fatty acids inhibits the formation of long-chain polyunsaturated fatty acids (LCPUFAs) from their precursors. LCPUFAs are important for both growth and the development of vision and the central nervous system early in life.
- **Allergy:** In an international study of asthma and allergies in childhood from 1998, the incidence of asthma, allergic cold and asthmatic eczema in children aged 13-14 years was investigated in 155 centres around the world. A link was found between the intake of trans fatty acids and these diseases.
- **Obesity:** Dietary fat and its relation to obesity has been a controversial issue for several years. Research with monkeys indicates that trans fats may increase weight gain and abdominal fat. A six-year experiment revealed that monkeys fed a trans-fat diet gained 7.2 per cent of their body weight, as compared to 1.8 per cent for monkeys on a mono-unsaturated fat diet.[1]

identified as genetically predisposed to cardiovascular diseases (CVDs); the risk is compounded due to the consumption of vanaspati and trans fatty acids, which are especially bad for the heart as they reduce the amount of good cholesterol (for more on health impacts, (see Box: *What trans fats do to us*).

Moreover, consumers in India are largely unaware that cooking mediums used outside their kitchens, especially to make certain packaged foods, are packed with these unhealthy trans fats. Companies manufacturing these foods and edible oils make loud claims of using or producing 'healthy' oils. CSE researchers, therefore, started examining branded edible oils to understand what the industry meant by its claims.

3. What did the CSE study find?

CSE's Pollution Monitoring Lab tested 30 samples of branded edible oil widely available in the market. The total fatty acid profile (saturated and unsaturated) comprising 37 components and nine trans fats was analysed.

These samples comprised vegetable oils (21 samples) – soybean, sunflower, safflower, groundnut, mustard, coconut, olive, sesame oil, rice bran and palm oil; partially hydrogenated oils (seven samples), desi ghee (one sample) and butter (one sample). They were tested according to the internationally used methodology of the Association of Official Analytical Chemists (AOAC) for fatty acids analysis (Method 969.33 Fatty Acids in Oils and Fats).

The tests found that in all the vanaspati brands, trans fat levels were five to 12 times higher than the world's only standard for trans fats in oil, set in Denmark, at 2 per cent of the total oil.

The level ranged from 23.7 per cent in the case of 'Panghat' (a Mawana Sugar brand), to the lowest level of 3.73 per cent in desi ghee of Milk Foods Ltd and Amul butter (see Graph: *Trans fats of the land*).

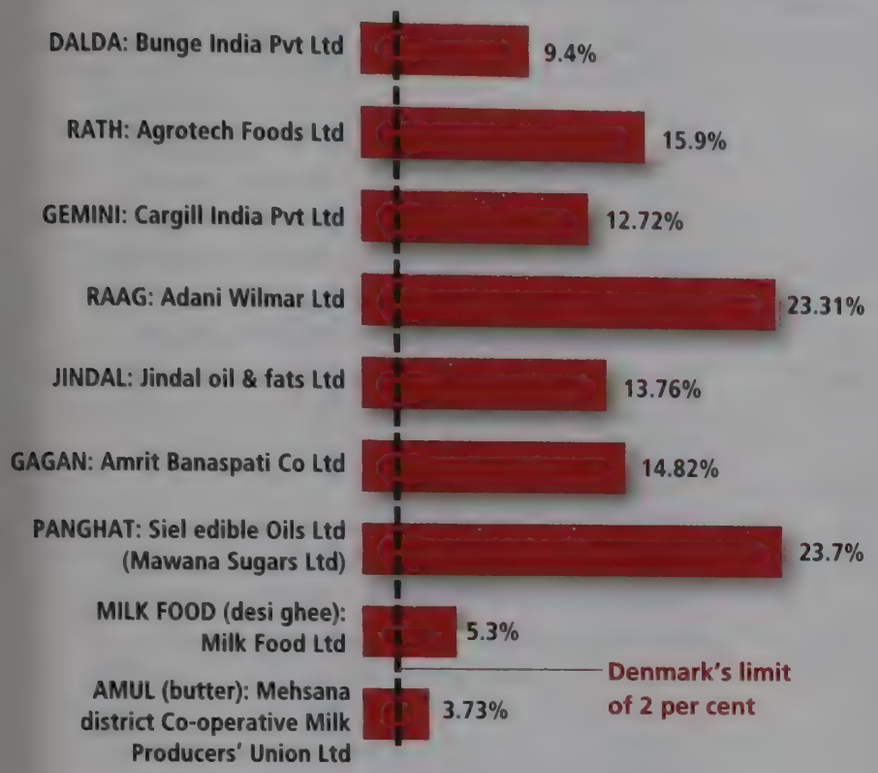
4. Does India have regulations for trans fats in cooking oil?

The edible oil industry is regulated under different standards:

- The **Food Safety and Standards Act (FSSA), 2006,** which became operational in August 2011, has

TRANS FATS OF THE LAND

Levels ranged 9-24 per cent – 5-12 times the standard in Denmark



Source: *Down To Earth*, February 15, 2009

taken the rules regulating the edible oil from its predecessor, the Prevention of Food Adulteration Act, 1956. The Act, under Schedule I of the regulation 4.1.9, specifies standards of edible oil and vanaspati giving broad specifications for different oils – cottonseed, coconut, groundnut, linseed, *mahua*, rapeseed, olive, sunflower etc. It includes standards for blended vegetable oils, which allow different oils to be blended and sold. Under this standard, companies can mix any quantity of any 'harmless' vegetable oil in their brand and can vary it as well.

- There are no standards for the cut-off of trans fats in the FSSA, 2006. The Act, however, provides for the labeling of trans fats, which has been included in a notification introduced by the ministry of health and family welfare in September 2008 on labeling for nutrition and health claims. It says only those products where trans fat is less than 0.2 gram (g) per serving of food should claim to be *trans fat-free*. In case a company makes nutrition or health claims on edible oil, then it is required to provide information on its package about the amount or the type of fatty acids including cholesterol, saturated fatty acids, MUFA (monounsaturated fatty acids), PUFA and trans fats.
- **Bureau of Indian Standards** lays down different specifications for edible oils and vanaspati, but has no standards for trans fats.
- **AGMARK:** This is a voluntary standard for vegetable oils and vanaspati governed by the Directorate of Marketing and Inspection of the Union ministry of agriculture, as per the Agricultural Produce Grading and Marking Act (1937). Blended edible vegetable oils and fat spreads are compulsorily required to be certified under AGMARK. But it does not have any standards for fatty acids or trans fats.

5. *How can companies make oil without trans fatty acids?*

There are options to reduce trans fats. Some of them are expensive, industry-patented and require a different technology. For example, Cargill has a line of all-purpose, semi-solid fats, TransEnd, which are made of a blend of canola oil and fully hydrogenated soybean oil, with less than 2 per cent trans fats. Bunge Oils has developed a proprietary method to hydrogenate edible oils with the production of less than 10 per cent trans fats during the process. But these are not available in India.

The other option is to use catalytic methods at relatively low temperatures. Another industrial way is to manipulate the fatty acid composition of oil seed using plant breeding and genetic engineering.

A simpler way is to change the oil – for instance, palm oil has lower trans fat potential. One can also move towards naturally saturated oils like coconut. Denmark, for example, found that 70 per cent of the products had substituted partially hydrogenated products with coconut and palm oil.

The problem is the Indian oil industry remains poorly regulated. It is allowed to mix oils. Technology allows it to get away, as it is possible to refine the oil and then doubly refine it so that we lose its feel and taste.

6. *What are the global regulations for trans fats?*

- **World Health Organization (WHO)** has recommended that governments around the world phase out partially hydrogenated oils if trans fat labeling alone does not lead to significant reductions. WHO also recommends that the trans fatty acids consumption should be less than 1 per cent of the total daily energy intake.

- **Denmark** became the first country to introduce laws strictly regulating the sale of many foods containing trans fats – a move which effectively bans partially hydrogenated oils. In March 2003, following notification in 2002, the Danish food authorities, on the ground that the measure was justified on public health grounds and was aiming at minimising the

risk of cardiovascular diseases, adopted a legislation which introduced (with effect from June 1, 2003) a limit on the level of trans fatty acids.

The following provisions are laid down pursuant to section 13, section 55(2) and section 78(3) of Act No. 471 of July 1, 1998 on foods (the Danish Food Act):

> *"A. From 1 June 2003, the content of trans fatty acids in the oils and fats covered by this Executive Order shall not exceed 2 grams per 100 grams of oil or fat, but see sub-article (2).*
>
> *B. From 1 June 2003 to 31 December 2003, the content of trans fatty acids in the oils and fats covered by this Executive Order which are part of processed foods in which food ingredients other than oils and fats are also contained, and which are manufactured in the food industry, the retail trade, catering businesses, restaurants, institutions, bakeries etc. may, however, be up to 5 g per 100 of oil or fat. From 1st January 2004, <2% TFAs are permitted in oils and fats used in both local and imported processed foods.*
>
> *C. In products claimed to be "free of trans fatty acids", the content of trans fatty acids shall be less than 1 gram per 100 grams of the individual oil or the individual fat in the finished product. Trans fatty acids shall be defined as the sum of all isomeric fatty acids with 14, 16, 18, 20 and 22 carbon atoms and one or more trans double bonds, i.e. C14:1, C16:1, C18:1, C18:2, C18:3, C20:1, C20:2, C22:1, C22:2 trans isomeric fatty acids, but only polyunsaturated fatty acids with methylene-interrupted double bonds.*[1]

- **Canada:** In January 2003, changes were made to the nutrition information in the country's Food and Drug Regulations. These required compliance by December 12, 2005 for large manufacturers, and by December 12, 2007 for small manufacturers. If claims are made (such as of reduced TFA levels), immediate compliance is required. Under its regulation, all vegetable oils and spreadable margarines must limit trans fat content to 2 per cent of total fat content. A limit of 5 per cent trans fat of total fat content in all products sold to consumers has been set.

- **USA:** On July 11, 2003, the US Food and Drug Administration (FDA) published a final rule in the Federal Register that amended its regulations on food labeling to require trans fatty acids be declared in the nutrition label of conventional foods and dietary supplements (68FR 41434) (effective from January 1, 2006). In August 2003, the FDA issued a detailed guidance document on interpretation of the regulations, titled 'Guidance for Industry: Food labeling: Trans Fatty acids in Nutrition labeling, Nutrient content Claims, and Health Claims'. In April 2004, the FDA Advisory Committee recommended that TFA intake be reduced to "...<1% of energy intake..." Subsequently (January 2006), the FDA required TFA levels also to be included on food labels, with percentage of saturated fat equal to the sum of saturated and trans fatty acids.

  The FDA decided not to distinguish between industrially produced TFAs and those derived from rumen hydrogenation; thus, dairy products must be labeled with TFA levels. This rule took effect on January 1, 2006.

  Trans fatty acids should be listed as "trans fat" or "trans" on a separate line under the listing of saturated fats in the nutrition label. Trans fat content must be expressed as grams per serving to the nearest 0.5 g increment below 5 g and to the nearest gram above 5 g.

  If a serving contains less than 0.5 g per serving, it can be labeled as '0' gram per serving or trans fat-free. Foods containing greater than 4 g saturated+ trans fatty acids cannot carry a health claim.[2]

- **European Union:** The European Food Safety Authority was asked to produce a scientific opinion on trans fats (European Food Safety Authority 2004). Declarations of the amount of TFA in a food are subject to the rules on nutrition labeling harmonized at the EU level. Nutrition labeling is voluntary unless a nutrition claim is made. Separate identification of the amount of TFA, as a component of the total fat content of the food is only required if a TFA nutrition claim is made. The commission has announced that the directive on nutrition labeling will soon be amended. In the UK and many other European countries, the situation is complicated. Although there is no specific requirement for labeling of trans fats on food labels, some manufacturers have started to do so voluntarily.

- **Australia and New Zealand:** Mandatory TFA labeling was considered during a comprehensive review of the Food Standards Code in 1999-2002; however, the Food Standards Australia New Zealand (FSANZ) and its precursor decided not to mandate labeling of TFAs as it was believed that TFA consumption was relatively low; and that similar reductions in saturated fat intake would be more likely to have a greater impact. TFA contents are required on labels only if a nutrition claim is made

with respect to cholesterol, saturated or unsaturated fatty acids or TFAs. Voluntary labeling is permitted and many vegetable oil spread manufacturers include TFA levels on labels. The Australian Heart Foundation recommends that saturated fats and TFAs should be 8 per cent of the total energy intake.

7. What has the Indian government done to set standards for trans fats?

Currently, India only has labeling laws on trans fats. The unanimous opinion is that food containing trans fats should be banned in the country or strictly limited in the manufacture of oil. But the government continues to drag its feet.

Discussions on setting TFA standards began way back in September 24, 2004. The Union health ministry's Oil and Fats Sub-Committee took up the matter of trans fatty acid in vanaspati. The sub-committee noted: trans fatty acids are health hazards; more harmful than saturated fats; and a 10-15 per cent limit may be safe. Based on this, the sub-committee requested the chairperson to prepare a paper.

On August 25, 2006 discussions resumed on the note prepared by the chair. But there was opposition. The director (vanaspati) of the government of India took the position that if standards are set many manufacturing units would not be able to meet the requirements; also that the number of samples tested were too small to fix the level of trans fats content. It is agreed that directorate of vanaspati would collect more data and submit it to the sub-committee.

On April 16, 2007, the sub-committee informed the Central Committee for Food Standards (CCFS) about its recommendation to set a limit of 15 per cent trans fats content in vanaspati. But again, there is resistance. The CCFS says that there isn't enough data to lay down limits. "The matter should be thoroughly debated by the sub-committee and then specific recommendations made," it says.

On January 7, 2008, the sub-committee met again. It discussed that the issue is gaining urgency, "in the light of growing presence of multinationals in the fast food business who have taken steps to limit trans fats in their products in western countries, but will take advantage of the lax regulatory control in the country". This will prove detrimental to our children, who are prone to obesity and their resultant diseases like diabetes and heart problems. A three-phase introduction of standards is recommended.

On February 18, 2008, the CCFS agreed to endorse the recommendation. It even asked for the standard to be set urgently, but called for data from the oil industry. This is where the matter ended.

8. What are the sticking points in setting the standard?

There are two central issues that dominate the discussion on trans fats. First is the limit of TFA in vanaspati/partially hydrogenated vegetable oil which is proposed to be fixed at 10 per cent and brought down to 5 per cent in three years. According to the WHO, in order to reduce the risk of cardiovascular diseases, not more than 1 per cent of the total energy should be derived from trans fatty acids. Similarly, the WHO says that not more than 10 per cent of the total energy in case of a healthy person and 7 per cent for those with cardiac problems should be derived from saturated fatty acids.

According to a risk assessment report prepared by the National Institute of Nutrition (NIN) on trans fatty acids in Indian diets, the fat consumption in rural and urban India is 20 g and 30 g per day, respectively. If a 10 per cent trans fatty acid level is permitted in vanaspati/PHVO, a person consuming 2,000 kcal will derive 0.9 and 1.35 per cent energy from the trans fatty acids. This shows that even at 10 per cent trans fatty acid level, vanaspati/PHVO will pose a risk to the urban consumer as it exceeds the 1 per cent energy limits to be derived for trans fatty acids as recommended by the WHO.

The second issue is that of the melting point. Trans fats are produced due to partial hydrogenation of oil that allows them to remain in a semi-solid state. Complete hydrogenation of the oil will lead to total removal of trans fats and will solidify the oil. The current melting point limit is set to 31°C to 41°C.

The vanaspati industry wants an increase in the melting point of its final product to reduce trans fats. But it is not so simple. Studies by the NIN have shown that a higher melting point increases the level of cholesterol in serum. Other studies have said vanaspati at a high melting point is difficult to digest. Also, there is a standard for the melting point, set by Agmark, at 31-41°C.

Oil major and multinational food company Bunge has proposed that the melting point of vanaspati should be increased to 44°C to carry out complete hydrogenation to reduce trans fats content. The Confederation of Indian Industry is arguing that if the melting point was increased to 45°C, trans fats level of 1 per cent was possible.

Caving in due to the pressure, the FSSAI has proposed to increase the melting point limit to 51°C or remove it all together.

Keeping the limit at 51°C does not solve the purpose because the industries do not prefer to hydrogenate at this temperature, as it leads to oil becoming too solidified which is not desirable to the

DRAFT REMAINS A DRAFT: PROPOSED REGULATIONS ON TFA

A. Level of TFA in vanaspati be fixed at the level of 10 per cent maximum and brought down to 5 per cent in three years. A phasing in period may be given to industry after the date of notification. A detailed review of the impact of the measure should be carried out by the end of three years to decide whether further reduction can be attempted.

B. There are no substantial studies on detrimental effects of raising the melting point of vanaspati from current level of 410 to 510 or to remove the melting point limit altogether. On the other hand, it has been argued that high melting point has no deleterious effect on health or digestion. Nor do Codex standards prescribe an upper limit on melting point. There is a need to carry out studies for documenting the effects of raising/removing the melting point limit on digestion and overall health. It is, therefore, proposed that status quo be maintained presently, i.e. the melting point of 41°C be retained or it be raised only to the extent that would facilitate bringing down the TFA level to the above limits.

C. There is also a need to look into the feasibility of laying down the limits of saturated fatty acids (SFAs) in vanaspati and other fats. This is being thought of because if the melting point is raised, it will lead to increase in saturation of partially hydrogenated vegetable oils. The WHO had recommended that not more than 1 per cent and not more than 10 per cent of energy in diet be derived from TFAs and SFAs, respectively.

D. Enzymatic esterification for production of vanaspati for regulating trans fatty acids can be considered. But being a costly alternative, it may take time for implementation.

E. As regards permitting the use of palm stearin in vanaspati, it also needs to be looked into whether to allow its use or not as in our country, palm stearin is used in the soap industry. However, there are no safety issues regarding its use and it is allowed in Codex with melting point of 44°C-52°C. Palm stearin, after interstratification with another liquid oil like rice bran oil, mustard oil, sunflower oil or soybean oil in appropriate ratio, is shown to produce shortening similar to vanaspati with comparable functional properties; the product is completely free from trans fatty acids. However, palm stearin content may be permitted only in interstratified fat and not approved for blending of oils or to be used as such.

consumer. Increasing the melting point limit will increase the saturated fatty acids content in the oil, which will then produce more than 10 per cent energy and which is beyond WHO's acceptable limit.

Therefore it is up to the regulators to choose the lesser of two evils: trans fatty acids or saturated fatty acids. Both saturated fatty acids and trans fatty acids are detrimental to one's health. Saturated fatty acids are naturally occurring fatty acids found in fats that are derived from animal products such as meat, dairy and eggs. They are also found in some plant-based sources such as coconut, palm and palm kernel oils. Saturated fatty acids directly raise the bad cholesterol levels (LDL). Trans fatty acids, on the other hand, not only increase bad cholesterol (LDL), they also decrease the good cholesterol (HDL).

9. What has happened since the CSE study?

In 2009, after the release of the CSE report, discussions on trans fats 'heated' up again. In March 2009, the Oil and Fats Sub-committee of the ministry of health and family welfare met to discuss the matter. It agreed to set a limit of 10 per cent TFA in vanaspati, but with the condition that the levels would be brought down to 5 per cent in three years. In other words, if the standard had been set, India would have achieved near-global standard by 2012.

But this recommendation was watered down. In November 26, 2009, the newly formed Food Safety and Standards Authority convened and agreed to recommend the limit of 10 per cent TFA in vanaspati.

In January 2010, the NIN conducted a national consultation on trans fatty acids. The consultation concluded that TFA levels in vanaspati should be below 10 per cent. It also recommended that the existing regulations for melting point, which stands at 31°C to 41°C, should be removed. This will be in line with the Codex Alimentarius guidelines.

In June 2010, the FSSAI issued a draft Regulation of Trans Fatty Acids (TFAs) in Partially Hydrogenated Vegetable Oils (PHVOs). The draft requires the level of TFA to be fixed at 10 per cent maximum and brought down to 5 per cent in three years (see Box: *Draft remains a draft: Proposed regulations on TFA*). The proposal, says FSSAI, will "reduce daily intake of trans fats by Indian consumers of all age groups to 1-2 per cent of energy intake consistence with dietary recommendations, over a period of three years".[3] It is not clear how this conclusion is arrived at, given that the draft proposes higher limits aas well as allow greatering mixing of oil.

The draft remains a draft. But the FSSAI claims that its recommendations have been sent to the ministry for notification; the ministry pleads ignorance. In the meanwhile, our health is being compromised, each day. ●

4.5 CAFFEINE

When CSE tested for pesticides in carbonated beverages in 2003, it also brought to light the caffeine content in these beverages and their health impacts. At that time, 200 parts per million (ppm) or milligram per kilogram of caffeine was the accepted norm. After CSE's study and rigorous campaigning, the permitted caffeine content was brought down to 145 ppm in 2009.

Though carbonated beverages have a caffeine standard, there are no caffeine standards for so-called energy drinks. Energy drinks came to India in 2002; their manufacturers say that their products are not under any regulations in India and hence, do not need to comply with caffeine limits. The earlier food safety law, the Prevention of Food Adulteration Act, had stated that all drinks containing caffeine should follow the rules applicable to carbonated beverages. The 2006 Food Safety and Standards Act specifies that caffeine will not exceed 145 ppm in non-alcoholic carbonated beverages.

The energy drink segment is a Rs 250-crore market and is now growing at an annual rate of about 20 per cent. CSE tested eight brands of energy drinks to check for the caffeine content in them and found that over 60 per cent of the brands had more than 145 ppm – a clear violation of the law. It is now believed that the Food Safety and Standards Authority of India is working on setting norms for energy drinks. However, the Authority is contemplating to set it at 320 ppm, which is in violation of its own stand that more than 145 ppm is harmful to health! It is also not clear why caffeinated drinks should qualify as 'energy drinks'.

1. *What is caffeine?*

A methylated xanthine, caffeine is a mildly addictive stimulant drug used in soft drinks as a 'flavoring agent'. It is a naturally occurring substance found in the leaves, seeds or fruits of at least 63 plant species worldwide, including cocoa beans, kola nuts and tea leaves. However, artificial caffeine is used in soft drinks. The most commonly known sources of caffeine in our foods are coffee, tea, soft drinks and now, energy drinks.

2. *How or why did caffeine become such a cause for concern?*

The Joint Parliamentary Committee (JPC) which was set up following CSE's 2003 study exposing high levels of pesticide residues in soft drinks, opened a

can of worms – besides deciding on pesticide residue levels, it also initiated an inquiry into the caffeine content in carbonated beverages (see CSE's 2004 publication: *Poison vs nutrition*). CSE contested the claims of soft drink manufacturers that caffeine was added just as a "flavoring agent". It cited a study conducted by Johns Hopkins Medicine (Johns Hopkins Hospital) in the year 2000, which had found that caffeine could not be detected as a flavor in soft drinks – and its use was more to do with inducing addiction to the drink than flavor.[1]

CSE also cited studies to show that large amounts of caffeine consumption could lead to diseases and disorders such as insomnia, nervousness, anxiety, irritability, and deviation from the normal heart rate. A major concern about caffeine is that it increases the excretion of calcium in urine, which heightens the risk for osteoporosis in heavy caffeine consumers.

Some epidemiological studies correlate exposure to caffeine during pregnancy to the occurrence of congenital malformations, fatal growth retardation, miscarriages (spontaneous abortions), behavioral defects and maternal fertility problems. In 1981, the US Food and Drug Administration had issued an advisory warning that "pregnant women should avoid caffeine-containing foods and drugs, if possible, or consume them only sparingly".[2]

Alarmingly, a very large proportion of those ingesting caffeine through soft drinks are young people – and these same people are now exposed to caffeine intake through another form of beverages rapidly riding the popularity charts: energy drinks.

3. What are 'energy' drinks? Are they different from carbonated beverages?

'Energy' drinks are beverages that contain caffeine, taurine, vitamins, herbal supplements and sugar or sweeteners, and are marketed as products that can improve energy levels, stamina, athletic performance and powers of concentration. Some of the leading brands of energy drinks being sold in India are Red Bull, Coca Cola's Burn, XXX and Cloud 9.

These drinks are the fastest growing beverages in the US market. In 2011, sales were expected to top US $9 billion.[3] Half of the consumers of these drinks include children (less than 12 years old), adolescents (12-18 years old), and young adults (19-25 years old).[4]

Though there is no formal definition for energy drinks, carbonated water has been defined in the Food Safety and Standards Act, 2006: "Carbonated water means potable water impregnated with carbon dioxide under pressure and may contain any of the following singly or in combination. Sugar, liquid glucose, dextrose monohydrate, invert sugar, fructose, sorbitol, honey, fruit and vegetables extractives and permitted flavoring, coloring matter, preservatives, emulsifying and stabilizing agents, acids, (citric acid, fumaric acid, tartaric acid, phosphoric acid, lactic acid, ascorbic acid and malic acid), edible gums such as (guar, karaya, arabic, carobean, furcellaran, tragacanth, gum ghatti, edible gelatin, albumin, licorice and its derivatives) salts of sodium, calcium and magnesium, vitamins, caffeine not exceeding 145 ppm..."[5] Unlike carbonated drinks in which caffeine is capped at 145 mg/l, these so-called energy drinks contain 320 mg/l caffeine or more.

4. How are energy drinks different from sports drinks?

Products promoted as sports drinks, like PowerAde and Gatorade, have glucose and electrolytes that replenish body fluids during strenuous activities. However, health experts suggest that these too should be taken with caution, especially since they typically have high levels of sugar.

Energy drinks, on the other hand, do not have electrolytes in them. Instead, they have high sugar and caffeine content that give the initial rush, rapidly – but temporarily – increasing energy and endurance. A paper published in 2011 in *Pediatrics*, the journal of the American Academy of Pediatrics, states that energy drinks are not designed to hydrate the body.[6] In fact, energy drink companies have purposefully placed their products in proximity to sports drinks to create what we call "consumer confusion". People are buying and drinking energy drinks, thinking them to be as innocuous as sports drinks.[7]

Red Bull India Private Ltd advises athletes to consume Red Bull half an hour before training or competing. According to the company, those performing in intense sports events can consume one or two cans during the event or at half-time break with adequate water; it can also be consumed after an event to aid recovery, the company says.

In their communications with CSE, company officials stressed on the fact that Red Bull is not a fluid replacement and that a lot of water should be had with the beverage. But this 'piece of advice' is missing from the product's labels.

Also, energy drinks are being misused: there is a growing trend of consuming energy drinks with alcohol (see Box: *Deadly duo*).

5. How do energy drinks affect our health?

Energy drinks is a misnomer. Except for sugar, the other ingredients in these drinks do not provide

DEADLY DUO

Energy drinks and alcohol has become a much sought after combination by youngsters at pubs and bars – Red Bull-vodka combination is particularly popular. In India, 90 per cent of the sales of energy drinks is due to its consumption along with alcohol.

The caffeine in energy drinks is believed to counter the effects of alcohol by warding off drowsiness, but studies reveal otherwise. The combination of the two can impair cognitive functions and reduce symptoms of alcohol intoxication, including the depressant effects, thus increasing the probability of accidents and favoring the possibility of development of alcohol dependence.

Energy drinks mixed with alcohol affect other organs as well. Alcohol inhibits the breakdown of nutrients into usable products in the body by decreasing the secretion of digestive enzymes from the pancreas. Electrolyte disturbances, nausea, vomiting and heart irregularities can also occur from excessive consumption.

People with diabetes should carefully watch their intake of energy drinks with alcohol as the combination can lead to fluctuations in blood sugars and chances of hypoglycemia are high. Patients with hypertension could have dangerous fluctuations in blood pressure. Patients with heart disease should strictly avoid these drinks.

energy. Many people get a sugar rush because of the amount of sugar – about 13 teaspoonfuls in a 500 ml can.

The 'feeling' of energy comes from caffeine, which is a stimulant. Other ingredients like taurine, glucoronolactone and ginseng have benefits but their content in energy drinks is too low to deliver therapeutic benefits or have adverse effects, claims a study published in the *Mayo Clinic Proceedings*.[8]

Caffeine can improve attention, but it also increases blood pressure and sleep disturbances in children. After cessation in children who habitually consume caffeine, attention decreases and reaction time increases transiently. Similarly, reaction time has been shown to decrease as the dose of caffeine in children increases.[9]

The work by researchers at the University of Adelaide and Royal Adelaide Hospital shows that the popular drinks, used by young clubbers, exercise enthusiasts and sleep-deprived mothers, can also increase the stickiness of the blood and damages blood vessel function.

As obesity is becoming an epidemic, caloric increases from energy drink consumption become important. Additional calories may increase blood pressure, blood glucose levels, Body Mass Index (BMI), calcium deficiency, dental problems, depression, and lead to low self esteem. Sugar and caffeine may also synergistically increase postprandial hyperglycemia (high blood sugar after a meal), which is of concern for children with diabetes.

Early adolescence is the time of maximal calcium deposition in bone, and caffeine interferes with intestinal calcium absorption. It remains controversial whether caffeine itself has the most marked effect on bone acquisition during adolescence or whether replacement of milk intake by caffeinated beverages is the leading contributor.[10]

Such beverages also contain Vitamin B and stimulants like taurine, glucoronolactone and guarana to create the energy blend. There is not enough study to show the cumulative impact of caffeine on them and till such time that ample research is not done, children and adolescents should be discouraged from consuming energy drinks.

Acute health impacts including death, due to overdose of energy drinks, is now being reported from different parts of the world. But no such reports exist for India. In India hospitals do not track cases of caffeine and energy drink overdose (see Box: *Not a safe thing, after all*).

6. How much caffeine is ok?

Studies say that adults who consume low-to-moderate amounts of caffeine (1-3 mg/kg body weight/day or 12.5-100 mg/day) have exhibited improved exercise endurance, cognition and reaction time. However, these studies typically involve habitual caffeine consumers, and results reflect withdrawal-symptom reversal.

Consuming 4 to 12 mg/kg body weight/day of caffeine has been associated with undesirable symptoms, including anxiety and jitteryness. Headaches and fatigue, common withdrawal symptoms, can occur after short-term, high-dose use. Caffeine intoxication is a clinical syndrome of nervousness, irritability, anxiety, insomnia, tremor, tachycardia, palpitations, and upset stomach. Additional adverse effects include vomiting and abdominal pain, hypokalemia, hallucinations, increased intracranial pressure, cerebral edema, stroke, paralysis, rhabdomyolysis, altered consciousness, rigidity, seizures, arrhythmias, and death. Caffeine intakes of more than 300 mg/day have been associated with miscarriage and low birth weight.[11]

For adolescents and children, caffeine consumption should not exceed 100 mg/day and 2.5 mg/kg per day, respectively.[12] Whether the effects of caffeine in adults can be generalised to children remains unclear. In a study of 26 boys and 26 men, the same dose of caffeine affected blood pressures similarly, but heart rate was significantly lowered in boys, whereas there was no effect on the heart rate in men. Boys also exhibited increased motor activity and speech rates and decreased reaction times than men.[13]

7. Why did CSE do a study on energy drinks?

Currently, regulations exist only for the caffeine content in carbonated beverages. In 2009, the Central Committee on Food Standards (CCFS), a statutory committee of the ministry of health and family welfare, scaled down the levels of caffeine allowed in carbonated beverages from the earlier norm of 200 to 145 ppm. This scaling down was based on the advocacy work done by CSE.

However, when the Food Safety and Standards Authority of India (FSSAI) set out to decide on standards for energy drinks, it was tilting towards allowing 320 ppm of caffeine in energy drinks. CSE saw it as a complete violation of the government's own stand that levels of more than 145 ppm is harmful for health.

As the energy drink market is completely unregulated in the country, CSE wanted to see how many of the brands available surpassed the permissible caffeine standards for carbonated beverages.

8. What were the findings of the CSE study?

CSE tested samples of eight brands of energy drinks. These brands were chosen on the basis of the popularity of the energy drink brands. The aim was to check the standards the energy drinks were following.

The study found that except for three brands- XXX Rejuve and XXX Nicofix of the JMJ group and Cloud 9 of Goldwin Healthcare- all the brands exceeded 145 ppm. The highest amount of caffeine, 314.09 ppm, was found in Monster-Ripper (Monster Energy Ltd) and the average levels of caffeine were found to be at 213.58 ppm. The lowest levels of caffeine were found in XXX Rejuve (JMJ Group).

The study also found that the energy drink manufacturers – brands such as XXX Minus, Burn and Cloud 9 – flouted labeling rules. While XXX did not mention the caffeine levels, Cloud 9 did not mention that it had caffeine and so conveniently excluded the caffeine levels. The tests showed they had 153.30 ppm and 142.25 ppm caffeine, respectively.

NOT A SAFE THING, AFTER ALL

In January 2011, about 300 people in the US suffered health problems from excessive consumption of energy drinks like Red Bull and Monster; most of them were children and teenagers, as per the American Association of Poison Control Centers. Germany has tracked energy drink-related incidents since 2002. Reported outcomes include liver damage, kidney failure, respiratory disorders, agitation, seizures, psychotic conditions, rhabdomyolysis (a fatal disease that destroys skeletal muscles and is often accompanied by the secretion of myoglobin or muscle haemoglobin in urine), tachycardia, cardiac dysrhythmias, hypertension, heart failure, and death.

Ireland's poison center reported 17 energy drink-related adverse events including confusion, tachycardia, and seizures and two deaths between 1999 and 2005. New Zealand's poison center reported 20 energy drink/shot-related adverse events from 2005 to 2009; 12 cases were referred for treatment of vomiting, nausea, abdominal pain, jitteryness, racing heart, and agitation.

ENERGY DRINKS REPORT CARD

Sample (Brand owner)	Caffeine level detected (ppm)	Caffeine level on the label (ppm)	Percentage deviation from the claim on the label	Number of times above PFA standard of 145 ppm
XXX Rejuve (JMJ Group)	117.14	100.00	17.14	within limit
XXX Nicofix (JMJ Group)	121.71	100.00	21.71	within limit
Red Bull (Red Bull India Pvt. Ltd.)	310.08	320.00	within Limit	2.14
Burn (Hindustan Coca Cola Beverages Pvt. Ltd.)	291.73	300.00	within Limit	2.01
XXX Minus (JMJ Group)	153.30	Not given	--	1.06
Cloud 9 (Goldwin Healthcare)	142.25	Not given	--	within limit
Monster-Ripper (Monster Energy Ltd)	314.09	300.00	4.69	2.16
Tzinga (Hector Beverages Pvt. Ltd.)	258.37	300.00	within Limit	1.78

Source: *Down To Earth*, July 31, 2011

The warning that lactating mothers should not consume energy drinks is missing from Cloud 9, Burn and XXX Minus labels. The label on XXX Minus was not legible. In the labels, there is no mention of the quantity of energy drinks that should be consumed.

Energy drink manufacturers pitch their products claiming that their caffeine content is equivalent to that of an average cup of coffee. This claim is made at 310.08 ppm of caffeine (Red Bull) and also at 258.37 ppm (Tzinga). This is misleading. A normal serving size of coffee is 100 ml and contains about 24-48 mg of caffeine, while energy drinks come in 250 ml cans and contain 80 mg of caffeine.

9. *What are the global rules and regulations for energy drinks?*

Countries like Denmark, Uruguay and Turkey have banned energy drinks altogether while Sweden has banned its sale among children. In Norway, energy drinks can only be sold in pharmacies; Australia too has proposed the same. In Finland, drinks that contain more than 150 mg/l of caffeine must be labeled "high caffeine content" as well as "not recommended for children, pregnant women, or people sensitive to caffeine". Energy drinks must also state the maximum amount to be used daily.

The German Federal Institute for Risk Assessment recommends that shots of energy drinks be banned because of the high risk of overdose. Ireland is reviewing the safety of energy drinks; its food safety board has recommended that energy drinks be labeled as unsuitable for children below 16 years and that a ban be placed on the promotion of Red Bull in sporting events and in combination with alcohol.

The Argentine senate is planning to propose a ban on energy drinks in nightclubs. The French had imposed a ban on Red Bull but lifted it after a report by the European Food Safety Authority (EFSA). The EFSA requires beverages with more than 150 ppm of caffeine to be labeled as "high caffeine content". Canada requires warning labels, recommends a maximum daily consumption of caffeine and advises against mixing of energy drinks with alcohol till there are ample studies.[14]

10. *How does India regulate energy drinks?*

When the energy drink Red Bull entered India in 2002, it fell in the category of proprietary foods as these products had no standards under the Prevention of Food Adulteration (PFA) Act of 1954. The PFA did try to introduce changes in the proprietary food segment and that would have brought energy drinks under the carbonated beverages segment; but the energy drink industry has managed to wriggle out of it and leave behind a legal case.

Energy drinks made news in 2005 when the Central Food Laboratory (CFL) at Mysore, under the direction of the customs authority at Chennai, tested Red Bull as a carbonated beverage and seized its consignment at the Chennai port as the drink did not conform to the standards prescribed by PFA for carbonated beverages, for which a caffeine cap of 200 ppm was mandated; Red Bull's caffeine content was about 320 ppm.

There was lack of clarity on the status of the drinks between the authorities – the PFA authority at the Centre had registered the beverage as a proprietary product (to which PFA Act rules did not apply), while the state food authorities conducted tests keeping in view norms prescribed for carbonated beverages.

Red Bull went into litigation. It challenged the findings of CFL and the seizure of the consignment in the Madras High Court. It pleaded that its product

should be treated a proprietary product, not carbonated beverage. The court ruled in favour of Red Bull, but CFL continued testing energy drinks as carbonated beverage. The energy drink giant dragged CFL to court on charges of contempt of court.

Red Bull was in trouble again when the regulation for proprietary food was amended in September 2008. The change in Rule 37-A (2) of the PFA Act stated that proprietary food products have to conform to the standards prescribed under various appendices of the PFA Act and Rules. The amendment implied that energy drinks, like carbonated beverages, would have to conform to the appendices of the Act, which capped caffeine limit at 200 ppm. Six months later, the caffeine limit in carbonated beverages was lowered to 145 ppm on the recommendations of the Central Committee for Food Standards.

After this amendment, the customs department at Chennai again seized consignments of energy drinks; tests by CFL showed energy drinks manufacturers were violating the rules. Red Bull again moved court, this time challenging the amendment and calling it unconstitutional.

The Food Safety and Standards Act includes the same definition of proprietary foods. The definition is as follows: "Proprietary and novel food means an article of food for which standards have not been specified but is not unsafe provided that such food does not contain any of the foods and ingredients prohibited under this Act and regulations made thereunder."

In addition, the Act includes a clear provision for caffeine limit in 'Beverages, non-alcoholic-carbonated (section 2.106). Red Bull, however, continues to operate outside this law by claiming it does not fall under this provision.

11. *What has been the FSSAI's role in drafting regulations for energy drinks?*

In 2010, the FSSAI had called for a risk assessment study on energy drinks. The study, done by the National Institute of Nutrition (NIN), Hyderabad, gave a clean chit to high caffeine levels in energy drinks. The study said that as the current consumption of caffeine in India was low, there was scope for more consumption.

But the NIN report is largely based on literature reviews. There is no sample study that has recorded daily intake of caffeine by people and the impact any additional intake may have. By estimating consumption on a per capita basis, which is quite low considering India's billion plus population, the NIN report has actually discounted the adverse impacts of energy drinks on a large caffeine-consuming section of the population.

A technical committee of the FSSAI, which reviewed the NIN report, made the following recommendations:

- Instead of calling these products energy drinks, the category should be called 'caffeinated beverages' – the term energy drinks may give an impression that these drinks can be taken to boost energy.
- Caffeine limits in energy drinks should be more than 145 ppm and less than 320 ppm.
- No health/functional claims can be made on caffeinated beverages unless specific approval is granted.
- No vitamins and nutritive claims should be permitted.

CUP OF COFFEE vs RED BULL CAN

	Quantity of serving (ml)	Quantity of caffeine (mg)	Caffeine (ppm)
Brewed coffee	100 (approximately)	24-48	240-480
Instant coffee	100 (approximately)	28	280
Green tea	100 (approximately)	8	80
Black tea	100 (approximately)	18	180
Red Bull	250	80	320
Burn	250	80	320

Notes: *measurements converted from ounce- 8 oz is approx 230 ml, In India coffee servings are small, about 100 ml
Source: coffeetea.about.com/library/blcaffeine.htm, as viewed on October 15, 2010

- The product should be packed in 250 ml containers.
- The companies should declare that daily consumption "should not exceed more than two cans".

But these recommendations have a long way to go – they have to be considered by the FSSAI, notified and then sent to the Union ministry of health and family welfare.

12. What has been CSE's response to the FSSAI's recommendation?

It is clear that there is no need for energy drinks in our diet: they do not add to the health or the energy levels in our bodies. The FSSAI has accepted the recommendations that were made by the National Institute of Nutrition (NIN) which stated that the current consumption of caffeine in the country is very low.

But the NIN report is largely based on literature review. There is no sample study to observe and record the daily intake of caffeine and the impact of additional intake through energy drinks. By estimating a per capita consumption of caffeine in a billion plus population in India it turns out to be very low. In fact the NIN report has actually discounted the adverse impact of energy drinks on a large caffeine consuming section of the population. It also discounts the intake of energy drink by a large section who mix it with alcohol.

Moreover, with existing caffeine sources such as coffee, tea and soft drinks, there is enough caffeine being consumed in India. Countries like Denmark, Uruguay and Turkey have already banned energy drinks due to the risks they pose to the health of consumers. A large proportion of energy drinks are being misused and consumed with alcohol and this combination hampers cognitive functioning.

If energy drinks cannot be restricted, then they should at least be capped at 145 ppm. If the PFA considered 145 ppm of caffeine as a safe limit for carbonated beverages, there is no reason why 320 ppm should be allowed for energy drinks. The government is flouting the norms that it had set for carbonated beverages.

The NIN tried to defend its stand by stating that caffeine is added to the cola beverages as a flavouring substance and that its purpose is only to provide a technological function. CSE replied that though used as a flavouring agent, the standards for caffeine in soft drinks were based on total diet studies and risk analysis in the EU and the US. And as total diet study and risk analysis have been the cornerstone of the standard setting process, it is a 'pharmacologically active substance'.

NIN also cited a study by Nawrot *et al* which does a risk assessment of caffeine by studying its effect among the adult healthy population. This in itself raises the validity of the study for setting standards for energy drinks, especially since they are being consumed largely by teenagers and children.

13. What should the government do?

CSE strongly recommends that there is a need to restrict energy drinks and if at all energy drinks are to be allowed, the caffeine content in them should be capped at 145 ppm.

Strict labeling laws need to be in place; misleading labels should be made punishable. Many labels on energy drinks compare a can of the drink to a cup of coffee – this is absolutely misleading on two counts (see Table: *Cup of coffee vs Red Bull can*). One, in India, the average size of a coffee cup is not 250 ml. Two, since a cold beverage with such high levels of caffeine can be consumed faster than a hot cup of coffee or tea, the body does not get enough time to metabolise the caffeine. Moreover, while tea and coffee have natural caffeine, only artificial caffeine is used in energy drinks. The labels should also write about the side effects of high levels of caffeine and the recommended daily intake of caffeine. ●

ANNEXURE: THE CSE LAB

The Pollution Monitoring Laboratory (PML) is a state-of-the-art lab set up by CSE to monitor environmental pollution and food contamination.

The lab came up in the year 2000, largely in response to community requests for independent testing of pollution. In India, pollution monitoring is done either by government agencies (including the Central Pollution Control Board) or private labs. While government agencies are generally reluctant to entertain community requests for testing pollution, tests done by private agencies are expensive. There was, therefore, a genuine need to establish independent labs in the country to cater to the needs of communities.

CSE took up the challenge. The very first study conducted by the PML was on endosulfan pollution in Kasaragod in Kerala; the study was done following requests from the community affected by aerial spraying of the pesticide. It conclusively proved that endosulfan poisoning was widespread in Kasaragod and was responsible for a host of health problems that people were facing there. Finally, in 2011, after a decade of struggle, the use, sale and manufacture of the deadly pesticide was banned by the Supreme Court.

In 2003, the lab did two more seminal studies: it tested for pesticide contamination, first in bottled water, and later, in soft drinks. The findings on bottled water led to the setting of standards which were at par with the world's best. Following the bottled water study and what came of it, consumers across the country wanted to know if soft drinks also were contaminated – and CSE tested all the major brands.

This soft drink study created history. A Joint Parliamentary Committee (JPC), only the fourth in the history of independent India, was constituted to investigate whether the findings of the lab regarding pesticide residues in soft drinks were correct. Not only did CSE's study come clean in the JPC probe, it also laid the grounds for two new laws – the Food Safety and Standards Act, 2006 and the Pesticides Management Bill, 2008.

The JPC also recommended setting strict pesticide standards for carbonated beverages. Two years passed by, with the government making no moves to set standards. In 2006, the PML tested soft drinks once again, and found high levels of pesticides. This was followed by another round of face-off between CSE, the industry and the government. Finally, in August 2008, the Union health ministry notified the rules for maximum levels of pesticides in carbonated beverages, making them the world's first standards on pesticides in soft drinks.

The lab has grown over the years. From testing pesticides in food and environment, it has moved to testing a wide variety of other contaminants.

In 2009, for instance, it intervened in the much contested issue of the Bhopal gas disaster site: its independent tests of water and soil samples from the Union Carbide factory site and the surrounding area showed how the waste dumped inside the factory had contaminated the groundwater within a radius of 3 km around the site. The CSE study was used as a reference study to file a liability clause in US courts. The study was also used by the Union government to develop a decontamination plan.

In 2005, the lab tested for pesticide contamination in the blood of farmers in Punjab. It triggered the debate on cancer and pesticides in Punjab, which has now become a hot political issue with every party recognising the ill effects of pesticides on the health of the state's people.

In 2009-10, the lab moved on to household chemicals that affected our daily lives. Studies were done on the lead content in paints sold in India, and on the levels of phthalates present in toys made or sold in the country.

In the last few years, the lab has covered new grounds: food and nutrition, and growth promoters and hormones used for boosting production are new areas of interest. In 2009, the PML did a study on trans fats in cooking oil; the study showed that India had no rules for trans fats in oil. This was followed by a study on the use of antibiotics in honey both as growth promoters and for therapeutic purposes. The study also brought to light the lack of any standards on antibiotics in honey for the domestic market.

The PML also did a study on caffeine in energy drinks. Though caffeine standards for carbonated beverages is set at 145 ppm, energy drink manufacturers continue to sell their products at a much higher caffeine level of nearly 320 ppm. The study also looked at mislabeling and lack of regulations in the energy drinks market.

In 2012, the lab conducted a major study on the nutritional analysis of junk food, focusing on high

sugar, salt and fat content as well as the issue of labelling of packaged junk food. The study kicked off a debate on mislabeling of junk food, lack of accountability and the health impacts of such food.

The PML is an ISO 9001:2008 certified laboratory accredited by SWISO, CH-5610, Wohlen, Switzerland, for conducting pollution monitoring and scientific studies on environmental and food samples. It has qualified and experienced staff members who exercise analytical quality control (AQC) and follow Good Laboratory Practices.

The PML is equipped with modern equipment for monitoring and analysis of air, water and food contamination. These include gas chromatograph with mass spectrometer (GC-MS), gas chromatograph (GC) with ECD, NPD, FID and other detectors, high performance liquid chromatograph (HPLC), atomic absorption spectrometer (AAS), UV-VIS spectrophotometer, mercury analyser, respirable dust sampler, etc. Its primary objective is to undertake scientific studies to generate public awareness about food contamination and environmental pollution. It provides scientific services at nominal cost to communities which cannot obtain scientific evidence against polluters in their areas. The lab and its work are directed to use science to achieve environmentally sound and socially relevant public policy.

LAB STUDIES — 2001-2012

Study	Investigators	Consultants	Date of release
Analysis of samples from Padre village in Kasaragod district of Kerala for endosulfan residues	Dr Padma Vankar, Dr Rashmi Mishra Dr Sapna Johnson		February 21, 2001
Analysis of pesticide residues in bottled water (Delhi region)	Prof (Dr) H B Mathur, Dr Sapna Johnson Dr Rashmi Mishra, Avinash Kumar Bhupinder Singh		February 4, 2003
Analysis of pesticide residues in bottled water (Mumbai region)	Prof (Dr) H B Mathur, Dr Sapna Johnson Dr Rashmi Mishra, Avinash Kumar Bhupinder Singh		February 4, 2003
Analysis of pesticide residues in soft drinks – first study	Prof (Dr) H B Mathur, Dr Sapna Johnson Avinash Kumar		August 5, 2003
Analysis of pesticide residues in blood samples from villages of Bathinda and Ropar in Punjab	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal Dr Sapna Johnson, Dr Nirmali Saikia		June 7, 2005
Analysis of pesticide residues in soft drinks – second study	Dr Sapna Johnson, Dr Nirmali Saikia Avinash Kumar	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	August 2, 2006
Fatty acid profile of edible oil and fats in India	Dr Sapna Johnson, Dr Nirmali Saikia	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	February 3, 2009
Study to analyse the lead content in paints sold in India	Dr Sapna Johnson, Dr Nirmali Saikia Ramakant Sahu	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	August 17, 2009
Checking the contamination of soil and water inside and outside the Union Carbide India Limited plant in Bhopal	Dr Sapna Johnson, Dr Nimisha Jadon Ramakant Sahu, Clara Duca	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	December 1, 2009
Study to analyse phthalate content in toys sold in India	Dr Sapna Johnson Dr Nirmali Saikia	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	January 15, 2010
Study to examine antibiotic residues in honey sold in India	Dr Sapna Johnson Dr Nimisha Jadon	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	September 15, 2010
Analysis of caffeine content in energy drinks sold in India	Dr Sapna Johnson, Dr Nimisha Jadon Ramakant Sahu	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	July 18, 2011
Nutritional analysis of junk foods	Dr Sapna Johnson, Ramakant Sahu Poornima Saxena	Prof (Dr) H B Mathur Prof (Dr) H C Agarwal	March 30, 2012

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BOX: An epidemiological study

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The Centre for Science and Environment (CSE)
is a public interest research and advocacy organisation based in New Delhi.
The Centre researches into, lobbies for and communicates the urgency of
development that is both sustainable and equitable.

The scenario today demands using knowledge to bring about change.
In other words, working India's democracy. This is what we aim to do.

The challenge, we see, is two-pronged. One one hand, millions live
within a biomass-based subsistence economy, existing at the margins
of survival; the environment is their only natural asset.
But a degraded environment means stress on land, water
and forest resources for survival.
It means increasing destitution and poverty.

The opportunity to bring about change is enormous.
But it will need a commitment to reform — structural reform — in the way
we do business with local communities.

On the other hand, rapid industrialising is throwing up new problems: growing
toxification and a costly disease burden.
The answers will be in reinventing the growth model of the Western world
so that we can leapfrog technology choices and find new ways of building
wealth, which will not cost us the earth.
This is the challenge of the balance.

Our aim is to raise these concerns and to participate in seeking answers
and more importantly, in pushing for the answers to become policy
and then practice.
We do this through our research and by communicating
our understanding through our publications.
We call this knowledge-based activism.
We hope we will make a difference.